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THIRTY-NINE EXTRAMEDULLARY TUMORS OF THE SPINAL CORD*

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FOREWORD

AT first thought one might be led to believe that a spinal cord tumor could be readily differentiated from other spinal cord lesions. If this be a general rule, there are many exceptions. We still make mistakes in diagnosis and at times in localization. The accumulation of evidence in a large series of tumors of known location and physical characteristics cannot but prove helpful. I have placed in the hands of Dr. K. Tamaki the records of 39 cases of extramedullary tumors of the spinal cord on my neurosurgical service, and he has prepared from these a statistical résumé of the important physical findings.

In reviewing my experience with tumors of the spinal cord when mistakes in diagnosis have been made, I have a very distinct impression that there was always an element of uncertainty in some particular. The history was not typical, the level of anesthesia was not distinct, perhaps there was no block, but whatever the reason may have been the operation was undertaken without the sense of assurance that comforts the surgeon.

There is another factor involved in these cases where no tumor was found. In the face of the otherwise unfavorable prognosis and the hopelessness of the situation, we have been persuaded to operate, perhaps too often, by the appeal of the patient. What else can be done?

I shall not discuss in this introduction the symptoms and signs of spinal cord tumor. The essayist has given to each its relative value and importance.

It seemed wise to limit the discussion to a particular type of tumor, rather than include all. The physical factors of the extramedullary tumors, their mode of expression, are quite different from those intramedullary. Since the extramedullary predominate in numbers, I selected a group of these for Dr. Tamaki's thesis.

CHARLES H. FRAZIER, M.D.

This contribution is an analysis of the clinical and pathological characteristics in a series of 39 extramedullary tumors, including 34 fibroblastomas, 4 fibromas, and one neuroma. Intramedullary tumors, tumors taking their origin from the vertebral column, sarcomas, metastatic carcinomas, and tumors of miscellaneous origin, such as angiomas, have not been included.

TABLE 1
AGE INCIDENCE

Age	*Fibroblastoma* (34)	*Fibroma* (4)	*Neuroma* (1)
15–19	3	0	0
20–29	1	2	0
30–39	8	1	1
40–49	12	1	0
50–59	4	0	0
60–65	6	0	0

* From the Neurosurgical Service and Laboratory of Dr. Charles H. Frazier in the Hospital of the University of Pennsylvania, Philadelphia.

An analysis of the age incidence shows that most of the cases of fibroblastoma, the most prevalent of these tumors, occur between the ages of thirty to fifty. Twenty of the 34 cases, or 59 per cent, fell within these age limits. For the entire series, the youngest patient was sixteen and the oldest sixty-five years of age.

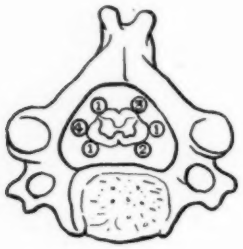


Fig. 1.

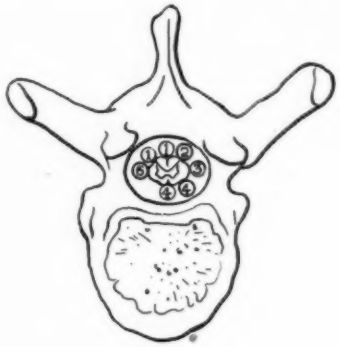


Fig. 2.

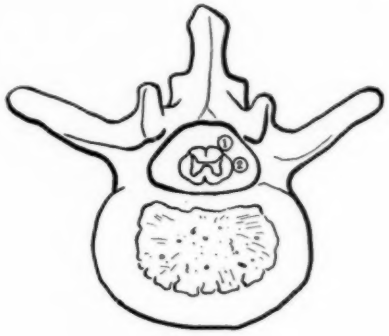


Fig. 3.

Fig. 1. Diagrammatic sketch of cervical cord to indicate incidence of tumors in relation to circumference of cervical cord.

Fig. 2. Diagrammatic sketch of thoracic cord, indicating number of tumors occurring at different positions of cord. There were no tumors on right anterolateral aspect of cord. Over rest of circumference, tumors were fairly uniformly distributed, except for a slight preponderance in right lateral aspect.

Fig. 3. Diagrammatic sketch of lumbar cord, indicating incidence of tumors in relation to circumference of lumbar cord.

ETIOLOGY

Sixty-nine per cent of the 39 tumors occurred in women. Although trauma has been cited frequently by many writers as an etiological factor (Oppenheim,[1] Bailey,[2] Learmonth,[3] and David[4]) its importance is still debated, and it is interesting to note that in this series there was a history of injury in but one case, a fibroblastoma, in which a blow on the back was followed about a year later by the onset of pain in the back.

RELATION TO CIRCUMFERENCE OF CORD

It is a well-known fact that fibroblastomas take their origin from the reflections of the arachnoid over the roots at their point of exit from the dural sac. Obviously, at the onset, the tumor occupies a position on the lateral aspect of the cord. Whether, as it grows, the tumor extends more posteriorly or anteriorly is not a matter of very great moment. Naturally one would expect the tumor to grow in the direction of least resistance, and no doubt the direction it takes would be influenced by its point of origin with relation to the dentate ligament and to the consistency and spread of the ligament. There is an extraordinary difference in the physical characteristics of this ligament. When the tumor is exposed at operation it is not always easy to define its exact position with relation to the cord circumference, that is, whether it is anterolateral or posterolateral.

The location of the tumor, as described at the operation, is represented diagrammatically in Figures 1, 2 and 3 or, if expressed in tabular form:

Table II

POSITION OF TUMOR IN RELATION TO CIRCUMFERENCE OF CORD

4	tumors were anterior
1	tumor was posterior
16	tumors were lateral
8	tumors were anterolateral
7	tumors were posterolateral
1	tumor was in the cauda equina
2	were not stated.

RELATION OF TUMOR TO MENINGES

Of 34 fibroblastomas, 4 were extradural, 24 were intradural, and 5 were dumbbell tumors (Table III). In his review of the literature and of the cases at the

Mayo Clinic, Learmonth[3] failed to find any extradural fibroblastoma, but later Hanlon[5] reported one case of extradural fibroblastoma from the same Clinic and Elsberg[6] reported 18 cases. Of the 24 cases of the intradural fibroblastomas, 14 were subdural, 7 subarachnoid, and 3 subpial. In most cases the tumor took its origin at the point of exit of the roots from the spinal canal. In some cases there was a prolongation of the tumor into the vertebral foramen. In 5 dumb-bell tumors, part of the tumor was outside and part inside the dura. Craig[7] in his series records 3 cases of dumb-bell shaped perineurial fibroblastomas, and two cases of perineurial fibroblastomas which developed within the spinal cord. In this series there were 3 fibroblastomas covered entirely by the pia mater and in one of the dumb-bell tumors one portion of the tumor was subpial, the other extradural. The single neuroma of this series was covered by the dura; it was 1.5 cm. long, 1 cm. broad and 0.75 cm. thick, was well encapsulated and apparently sprang from the anterior root.

Table III
Relation of Tumors to Meninges

Type of Tumor	Extradural	Intradural			Tumors having both extradural and intradural parts	Not stated	Total
		Subdural	Subarachnoid	Subpial			
Fibroblastoma	4	14	7	3	5	1	34
Fibroma	1	1	1	0	1	0	4
Neuroma	0	1	0	0	0	0	1
Total	5	16	8	3	6	1	39

There were 4 fibromas in the series, one an hour-glass tumor both extradural and intradural, one subdural, one extradural, and one, as in the case to be cited, subarachnoid.

Case 1. *Summary.* A patient in his fortieth year came to the Clinic eight months after the onset of his illness, with the characteristic sequence of symptoms, pain, paresthesia, paralysis. The tumor, a subarachnoid fibroma, was found and removed. Perfect recovery.

S. G., male, aged thirty-nine years (File No. 6580 N.S.) was referred to the Neurosurgical Service of the University Hospital by Dr. T. H. Weisenburg, September 15, 1925, with the following history:

In January, 1925, he began to have pain in the back; it was very sharp at times, worse at night, and often awakened him. Change of posture seemed to give him relief, as the pain was better when he got up and walked the floor. The pain has persisted to the present, though it has been less severe since July.

In March, 1925, numbness developed in both feet and slowly spread to the waist, at which level it has remained fixed for some weeks. His legs feel "tight" as though full of blood.

In April, 1925, he began to have difficulty in walking; his limbs grew progressively weaker until four weeks ago he was unable to walk unassisted. He feels as though he were walking on something soft. Before admission a provisional diagnosis of pachymeningitis (either syphilitic or tuberculous) had been made and he had received active antisyphilitic treatment, without effect.

Neurological Status. Motor Phenomena: There is partial paralysis of both lower extremities with some wasting of muscles. An occasional fibrillary tremor is seen. Gait is uncertain; he walks with wide base and watches feet.

Sensory Phenomena: Impairment of touch and tactile sense to the second interspace. Sense of pain more impaired on right side. Loss of sense of position of great toes. Vibratory sense impaired from hips down. (See chart, Fig. 4.)

Reflexes: Abdominal, absent. Patellar tendon, exaggerated. No pathological reflexes.

Cerebrospinal Fluid: Pressure 210 mm. Lymphocytes 7. Colloidal gold and Wassermann, negative. Spinal block. Fluid xanthrochromic.

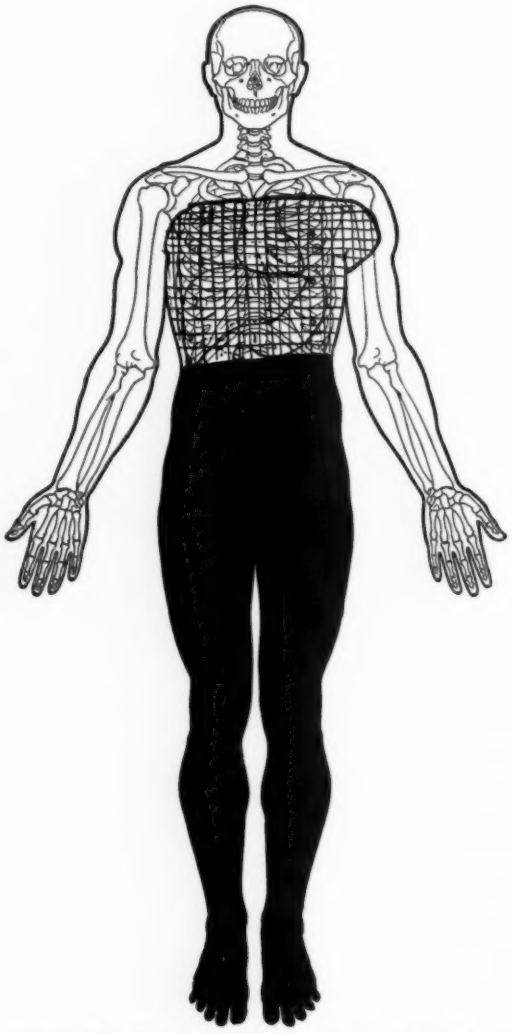


FIG. 4. Anesthesia chart, Case 1, showing loss of temperature, pain and tactile sensation (solid black); moderate impairment above.

Roentgenogram of spinal column, negative.

Operation: Laminectomy, September 23, 1925. Tumor found and removed. For location and physical characteristics of tumor see Figure 5.

Pathological Diagnosis: Fibroma.

Postoperative Note: Convalescence uneventful. Three weeks after operation motor power restored. October 1926, when last seen, examination essentially negative.

LEVEL INCIDENCE

It has long been known that the site of predilection for spinal cord tumors is in the lower cervical and the upper thoracic segments. It was pointed out by Frazier[8] some time ago that the preponderance of

FIG. 5. Drawing at operation, Case 1, for removal of fibroma.

incidence in the thoracic region might be due to the relatively greater length of that section of the cord. Whatever the predisposing factor may be, whether because of the greater mobility, of the greater exposure to injury, or whether there be embryological or anatomical factors, it is true that in any series the vast majority will be found between the fourth cervical and the sixth thoracic segments. In this series 31 per cent occupied the cervical region, 59 per cent the thoracic segments, and 10 per cent the lumbar segments or cauda equina (see Fig. 6).

As to the physical characteristics of the fibroblastomas and fibromas; there is in the majority of our cases a striking uniformity as to size and shape. This no doubt is due to the limitations to growth offered by the spinal canal. Obviously, the tumor can grow more readily in its vertical than in its transverse diameter, so that these tumors are usually much the shape of an olive, greater in their vertical than in their transverse diameter (Fig. 7). The average dimensions are 2–3 cm. in length, and 1.5 in breadth. There are, however, striking exceptions. Figure 8 is an illustration of a tumor of much larger dimensions exposed in the cervical canal, and under these circumstances one

finds an excavation by pressure atrophy of the bony wall of the vertebral canal. So, too, in an occasional case the tumor will be

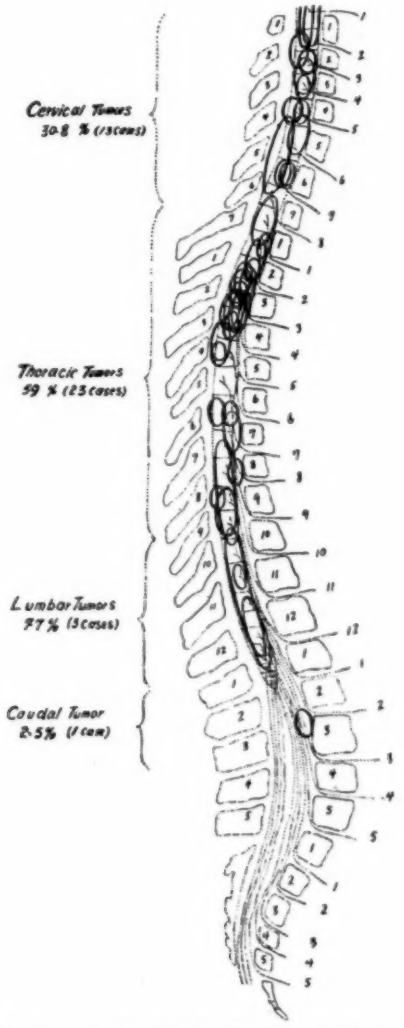


Fig. 6. Diagrammatic sketch of spinal cord seen in longitudinal section from side, to indicate position of spinal cord tumors in this series. Most of them, it will be noted, are in thoracic region.

three or four times longer than one of the usual dimensions (Fig. 9).

SYMPTOMATOLOGY

Pain. In the Neurosurgical Clinic of the University Hospital, more emphasis has been laid upon pain perhaps than elsewhere, but the statistics of the Clinic justify this position. Pain has been a conspicuous symptom in every case but one and, what is of still greater significance, was the first symptom in 82 per cent of the series. Of course, there are exceptions in this Clinic as in others.

The painless course of an extramedullary

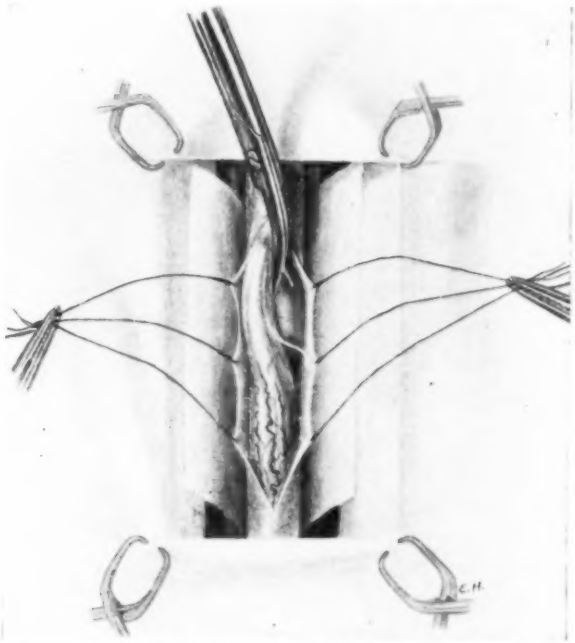


Fig. 7. Drawing in case of fibroblastoma to show usual olive-like shape of tumor, greater in vertical than in transverse diameter.

tumor must be acknowledged as unusual but there are cases on record (Bailey,[2] Ayer,[9] and Elsberg[10]). Even though, as in Sharpe's[11] case, the posterior roots were flattened by the tumor, the patient was without pain. So, too, in cases recorded by Collins and Marks[12] the posterior roots were stretched over the tumor, but the patient complained neither of pain nor of paresthesia throughout the entire course of the disease.

In the experience of this Clinic, pain, besides being almost universally the first symptom, is also the symptom of greatest significance; first, because of its value in determining the segmental level, and secondly, because the persistence of pain explains many mistakes in diagnosis.

The localizing value of pain was emphasized early by Frazier and Spiller.[13] In their opinion, pain is first observed at a more or less definitely localized point, almost invariably at a level corresponding

to the root or segment involved. Pain appears early and persists in the original area, although in the course of the disease to which pain is referred varies according to the level of the lesion and often according to whether it is due to root or cord tract

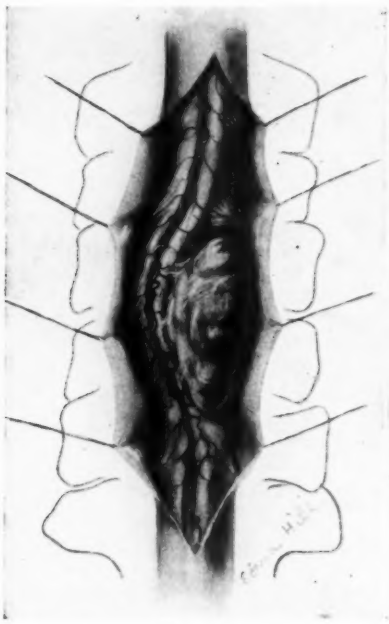


FIG. 8.

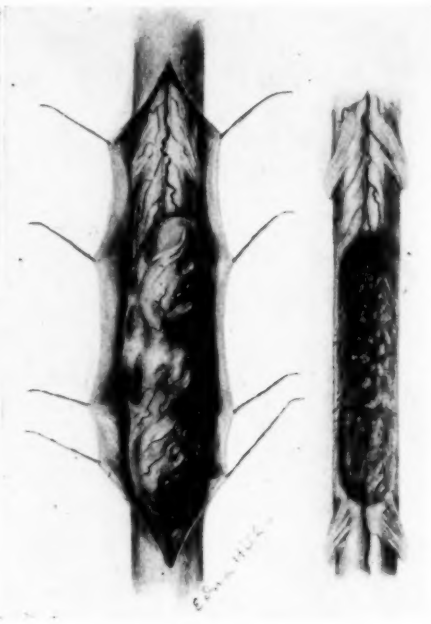


FIG. 9.

FIG. 8. Drawing in case of fibroblastoma, showing tumor of unusually large dimensions, causing pressure atrophy of bony wall of vertebral canal.

FIG. 9. Drawing in case of fibroblastoma, showing unusually elongated tumor.

it may be intermittent and its nature may vary. Its character varies somewhat according to whether it be due to involvement of root or cord segment. When of root origin the pain is often described as neuralgic, knife-like, boring and rheumatic. Pain resulting from pressure on the antero-lateral tracts of the cord has been noted as burning, cramp-like, drawing or as a dull ache. Of these, "cramp-like" was the term most often used; the "drawing" pain seemed to imply a rather advanced lesion and was associated always with other evidence of cord damage, almost invariably with involuntary contractions of the lower extremities.

Pain is aggravated by movements of the body and change of position, as in stooping, by jarring, by sneezing and coughing, the latter of great diagnostic significance. Many patients complain of inability to sleep because the lying posture seems to make the pain worse (see Case I). The zone involvement. When of root origin, obviously it is referred to the distribution of the root involved. On the other hand, when pain is due to cord tract involvement, it may be referred to the segment involved or to distant parts, as to the lower extremities. In this series of 39 cases pain *en distance* was recorded in 12 instances, in 4 cervical and 8 thoracic tumors. In all of these it was referred to the lower extremities and to both extremities in all but one instance. In one case it was referred to the foot contralateral to the lesion.

Obviously, at first, root pains are unilateral and often, though not always, referred to the terminal peripheral distribution as, for example, to some point on the abdomen or anterior aspect of the thorax (see Case III). With cervical tumors the pain may be felt in the back of the neck, in the side of the neck, in the shoulder or in the upper extremity; in thoracic tumors in the back, under the scapula, between the

shoulders, in the region of the kidney, in the sternum, in the precordium, under the costal margin or in the abdomen. In a tumor at the ninth thoracic segment, pain was referred to the right costal margin; a diagnosis of cholelithiasis had been made and the patient subjected to an abdominal operation before she was admitted to this Clinic. Similarly, in Beckman's[14] case, the pain was referred to the region of the gall bladder. In Kamman's[15] case of thoracic tumor there was excruciating pain along the intercostal nerve with a typical herpetic eruption.

In tumors involving the lumbar segments pain was referred to the lower back, the lower abdomen or lower extremity. In one case, there was a colicky pain in the bowel. In caudal tumors, pain was often felt in the lower back, especially in the small of the back, and from this point radiated to the leg. In some cases pain was unilateral in the beginning, later becoming bilateral, and in other cases it was bilateral from the beginning. Warrington[16] says that a pain, violent in nature, spontaneous or excited by change of position or by movement, is very characteristic of caudal tumors. Viets[17] found that the pain of caudal tumors is intensified by compression on the jugular vein.

Girdle Sensation. The "girdle" sensation is commonly expressed by the patient as a feeling of constriction around the chest or abdomen and Dandy[18] thinks it is the patient's interpretation of the sudden change from the normal to the disturbed sensation. It may or may not be painful. Girdle pains have been frequently discussed in literature, among others by Hunt and Woolsey[19] and by Castelli.[20] In this series girdle sensation was noted in 8 cases and always appeared late in the course of the disease. It was the third symptom in 4 cases, the fourth in 3 cases, and the fifth in one case, but never the first or the second symptom. The level to which the girdle sensation is referred is worthy of comment. In two cervical tumors, the girdle pain was felt in the abdomen. In six thoracic tumors the sensation was felt at various levels below the breasts. In 2 of these cases, in which the tumors were located one at the level of C. 8, T. 1, T. 2, and the other at T. 5, the girdle sensations were experienced around the abdomen. In the other 4 cases with a tumor at T.4, the girdle sensation was referred to a zone between breasts and umbilicus; with a tumor at T.4 and 5, to the lower ribs; with a tumor at T.4 and 5 around the chest; and with one at T.9, around the waist.

Of the other sensory disturbances, a word should be said of subjective numbness. At first this usually affects the contralateral and later the homolateral limb, when due to cord involvement, but when due to root invasion the numbness is homolateral. As a means of differentiating between intramedullary and extramedullary tumors it should be remembered that with the former the numbness spreads from above downward toward the periphery. The reverse is true of extramedullary tumors.

Paresthesia. As to its chronological significance, paresthesia, next to pain, is the most common symptom. The usual sequence is pain, paresthesia, paralysis. Paresthesias were present in 75 per cent of the cervical tumors, in 87 per cent of the thoracic tumors, and in every case of tumor in the lumbar segments and cauda equina. They were variously described as numbness, tingling, formication, pricking, burning, dryness, coldness, stinging, pins and needles, and a feeling as if a cold wet stocking had been applied to the limb (see Case 11). A sense of cold is said by Elsberg and Strauss[21] to be characteristic of a tumor of the cervical cord, but this type of paresthesia was found also in four of the thoracic tumors in this series.

Case 11. *Summary.* A tumor, both intradural and extradural, was found and removed from a patient with a history of pain, paresthesia and paralysis. She had a sensation of having cold wet stockings on both legs.

F. M. L., female, aged sixty years (File No. 7606), was admitted to the Neurosurgical

Service of the University Hospital October 2, 1925, complaining of inability to walk.

History: The patient was well until April, 1925, when she began to have lancinating pains in her legs at night, lasting but a few seconds, but recurring in rapid succession. *In the day time she had the sensation of having cold wet stockings on her legs.* Soon after this her legs began to feel stiff and in July she noticed difficulty in stepping over low railings. On admission, October 2, 1925, she could move her legs easily and walk a little, but now she has lost all power in her lower limbs.

Neurological Status: No involvement of the upper extremities.

Reflexes: Abdominal reflexes lost. Patellar and Achilles tendon reflexes exaggerated on both sides. Positive Babinski and sustained ankle clonus, right and left.

Sensation: (See charts, Figs. 10 and 11.) There is complete loss of sensation for touch, pain, temperature, position and pressure up to the fourth thoracic dermatome on the right and fifth thoracic dermatome on the left. There is tenderness to pressure over the spinous process of the third thoracic vertebra.

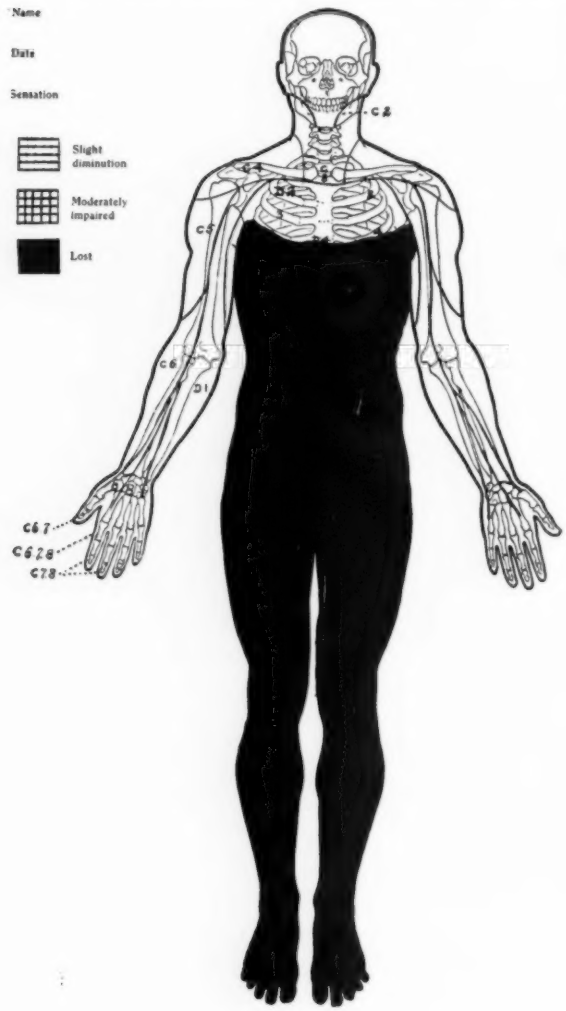


Fig. 10.

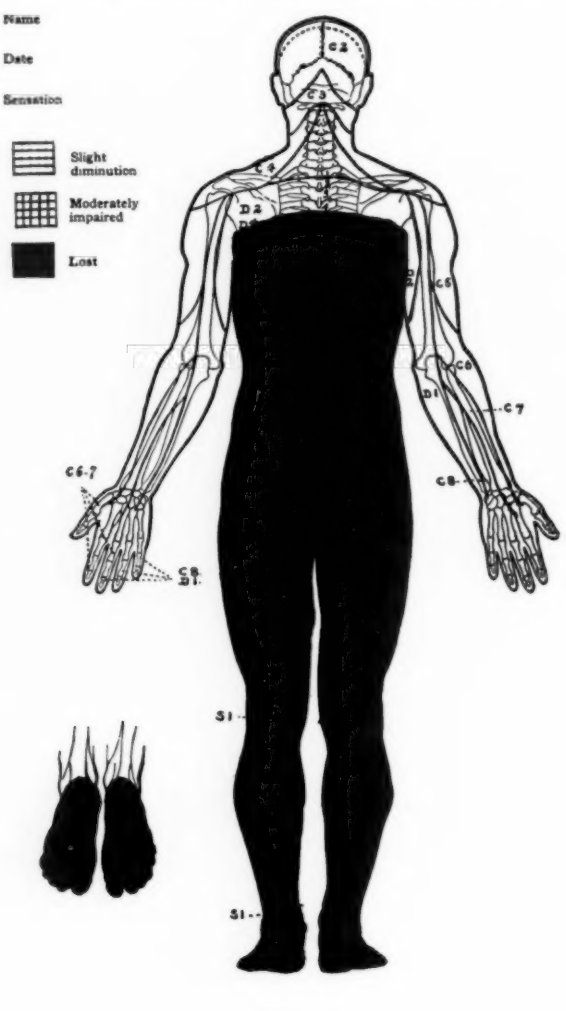


Fig. 11.

Figs. 10 and 11. Sensory charts, Case 11, showing loss of sense of touch, pain, temperature, pressure and position.

Motor Phenomena: Total paralysis of both lower extremities with the exception of slight movement in the left toes.

Cerebrospinal Fluid: Wassermann and colloidal gold tests negative. The Queckenstedt test showed a complete block.

Operation: Laminectomy, November 23, 1925, local and ether anesthesia. The tumor consisted of two portions, one intradural and one extradural. The intradural portion was subarachnoid and displaced the cord to the left. The extradural portion at the same level had

eroded, and at one point perforated, the bone. Both intradural and extradural portions were completely removed. (Fig. 12.)

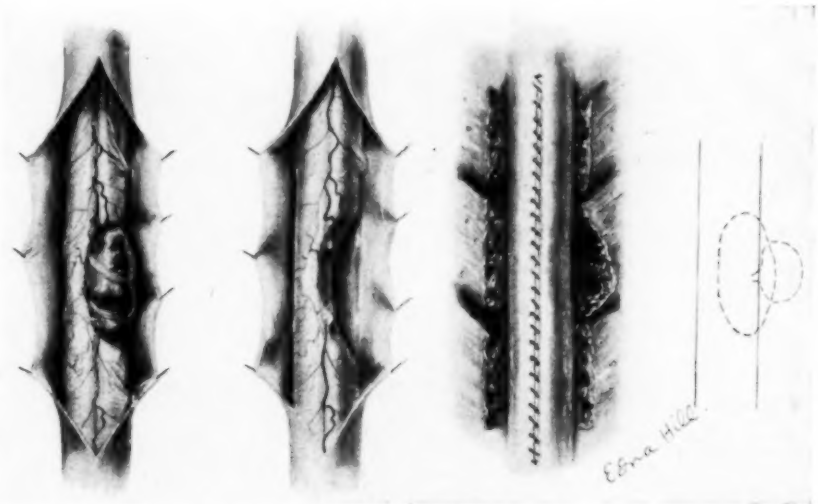


Fig. 12. Drawing at operation, Case 11, during removal of an intradural and extradural fibroblastoma.

Postoperative Note: Recovery from the operation was uncomplicated, and before discharge from the Hospital, January 27, 1926, the patient was able to take a few steps unassisted.

Pathological Diagnosis: Fibroblastoma.

Paresthesias are the result either of irritation of the nerve roots or of pressure on the sensory tracts. When due to root irritation, their localizing value is comparable to that of root pain, but in the majority of cases they are due to pressure on the sensory tracts. In such instances, the paresthesias are referred to areas below the level of the lesion. In each of the 9 cervical tumors, the paresthesias involved some part of the upper extremities. When the tumor was below T. 2, they were referred to the lower extremities or the trunk. At the outset the paresthesia is usually referred to the extremities, especially in their distal areas, such as the toes, the legs, the fingers, or the hands, but later it gradually spreads to the trunk.

The relation of the initial area of paresthesia to the position of the tumor on the circumference of the cord deserves consideration. There were 12 intradural extramedullary tumors on the *lateral* aspect of the cord; in 4 of these the initial area was homolateral, in 2 bilateral, while in 5 there were no paresthesias. In only one of the lateral tumors was the paresthesia contralateral. Of 5 intradural tumors on the *posterolateral* aspect of the cord, the initial area was bilateral in 2, homolateral in 3 cases, but contralateral in none. Of 9 intradural extramedullary tumors *on the anterolateral aspect* of the cord, the initial paresthesia was contralateral in 4, homolateral in 3, bilateral once, and absent once. Thus, it is apparent that paresthesias are contralateral almost exclusively in tumors on the anterolateral aspect of the cord. This might readily be accounted for by the approximation of the tumor in this location to the spinothalamic tracts. Our statistics, therefore, are not in accord with a prevalent belief that the initial area of paresthesia is contralateral in the majority of cases of intradural extramedullary tumor. In 4 extradural extramedullary tumors the initial area was bilateral in 2, homolateral in 2; in none was the distribution contralateral.

Motor Weakness. With respect to chronological sequence the predominance of sensory disturbances as the initial symptom has already been emphasized. In only 18 per cent was motor weakness the first symptom observed. It usually begins

with a subjective complaint of stiffness or weakness of some distal part of the extremities, such as the foot, leg or hand. The legs are most commonly affected, regardless of the level of the tumor. Its progress is usually gradual and from the periphery centralwards, as with the paresthesias. Motor weakness from the outset may be either bilateral or unilateral. When the tumor was exactly anterior or exactly posterior, the initial motor weakness was predominantly bilateral. However, when the tumor was anterolateral, posterolateral or lateral, the initial motor weakness might be unilateral and usually homolateral with the lesion. Of the 7 tumors on the *posterolateral* aspect of the cord, motor weakness at the onset was bilateral in 3 and unilateral in 4. Of 16 tumors *laterally* located, motor weakness was primarily unilateral in 10, bilateral in 6. Of 8 tumors on the *anterolateral* aspect of the cord, 5 exhibited unilateral and 3 bilateral motor weakness. Of unilateral motor disturbances, weakness was homolateral in but one case, an extradural tumor on the lateral aspect of the cord at the second lumbar segment.

As pressure on the cord continues, the unilateral weakness usually becomes bilateral, although in a few cases it may remain unilateral. In the majority of upper cervical tumors the homolateral upper extremity is first affected and later all the extremities. In most of the lower cervical tumors the lower extremities were first involved. In 2 cases of lower cervical tumors paralysis was limited to the lower extremities throughout the entire illness.

Of the 23 cases of thoracic tumor, 9 exhibited unilateral and 14 bilateral weakness from the beginning. When the motor involvement is primarily unilateral, it eventually becomes bilateral. In the case of a tumor at C.8 and T.1 and T.2, there was stiffness of the little finger of the right hand, in addition to bilateral weakness of the lower extremities. In another case in which the tumor involved T.3 and T.4, there was motor weakness of the left upper extremity, in addition to paralysis of both lower extremities. Obviously with tumors in the lumbar segments and in the cauda equina, the lower extremities alone were involved and in every instance both lower extremities.

Objective examination does not always corroborate the subjective sensation of weakness. For instance, there is often motor power remaining in the extremities, although the patient may complain of complete paralysis. The paralysis may be spastic or flaccid, the spastic type by far the most common. Flaccid paralysis was observed in one cervical tumor and also, in the case of a tumor of the cauda equina.

As noted, flaccid paralysis is rare with tumors located elsewhere than in the cauda equina, and when it occurs, it is usually associated with a rapid progression of symptoms. According to Starr[22] compression of a tumor upon the spinal cord produces at first spastic paralysis and later as the compression progresses the paralysis becomes flaccid. But the spastic paralysis produced by gradual pressure upon the cord remains spastic, even after the extremities have completely lost voluntary motion. Flaccid paralysis, produced by a slowly growing tumor, usually implies advanced damage to the cord, although Learmonth found in his series of cases that a patient who had suffered from complete flaccid paralysis recovered his motor power after operation. In Nattrass'[23] case, also, a patient who had suffered from flaccid paralysis for twelve months recovered power after the operation. There have been reported numerous cases of spinal cord tumor in which flaccid paralysis developed very suddenly after lumbar puncture. In Nonne's[24] case, the flaccid paralysis following lumbar puncture was due to a sudden hemorrhage into the tumor.

Sphincter Disturbance. In 26, or 67 per cent of the 39 cases, there was sphincter disturbance, either vesical or rectal, and in some cases both sphincters were in-

volved. Urinary disturbance was found in 59 per cent of the 39 cases, beginning as a rule with difficulty in starting the expulsive act. Some patients had urgency, while others complained of painful urination and, subsequently, either complete retention or complete incontinence. Even after incontinence has set in, a period of remission may occur. Since the mechanism of bladder control has to do with the sympathetic fibers, which leave the cord by the last two thoracic and first two lumbar roots, as well as from the second, third and fourth sacral segments, one would expect bladder complications to appear earlier in tumors of the lumbosacral region.

Rectal incontinence was found in 20 cases, or 51 per cent of the 39 cases, and constipation in 11 instances. In the majority of instances, sphincter disturbances appeared as the fourth or fifth symptom; in only 3 cases, as in the following (Case III), did it appear at the onset of the disease.

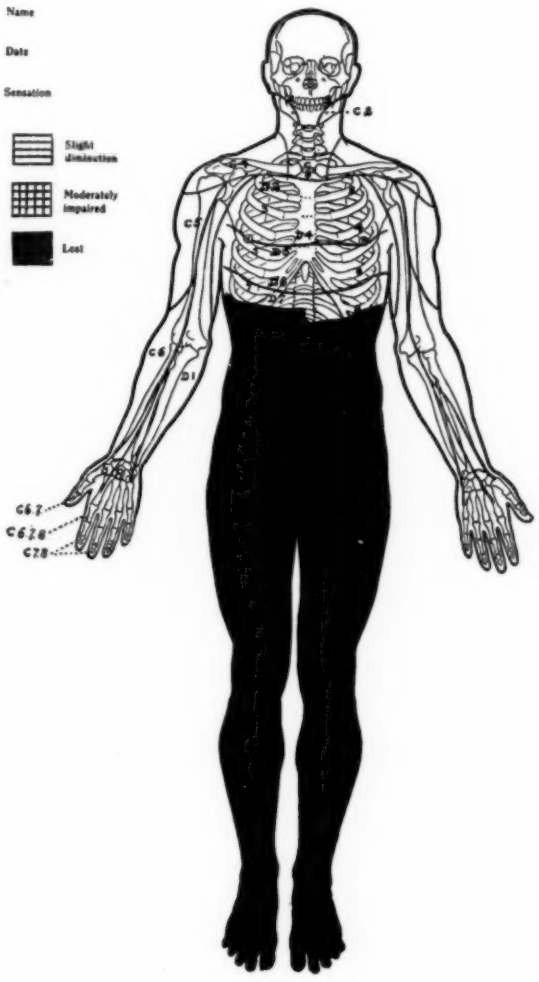


Fig. 13.

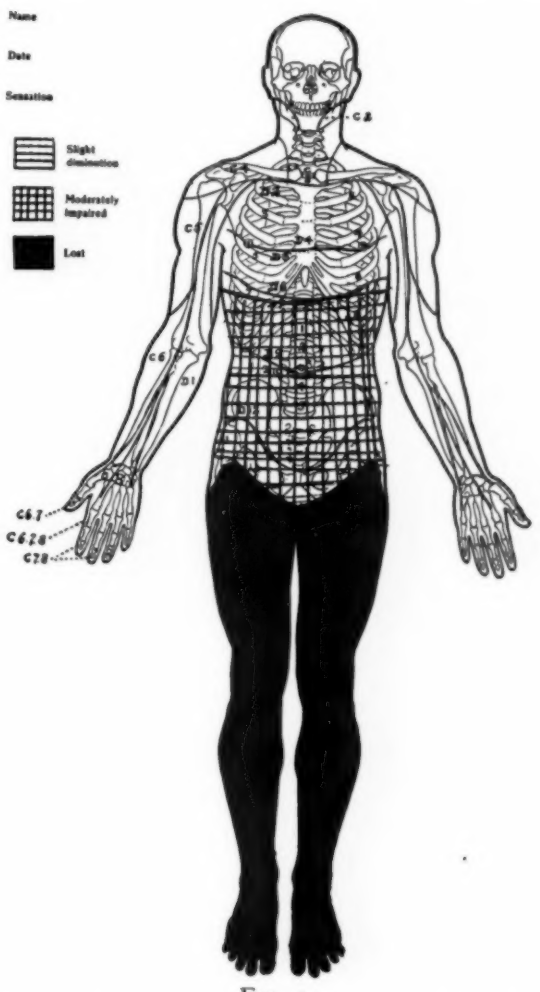


Fig. 14.

Figs. 13 and 14. Sensory charts, Case III, showing loss of sense of temperature and pain up to level of T. 7.

Case III. *Summary.* Almost simultaneously with the onset of the first symptom (stiffness of left knee) eighteen months before admission, the patient developed incontinence of bladder and bowel. Eight months after the removal of a perineurial fibroblastoma from the posterior aspect of the cord at the eighth thoracic segment, convalescence was well established.

Mrs. F. Van S., aged forty years (File No. 23156) was referred to the Neurosurgical Service of the University Hospital by Dr. William G. Spiller, November 12, 1931, with numbness and complete paralysis of both lower extremities and loss of bladder and bowel control.

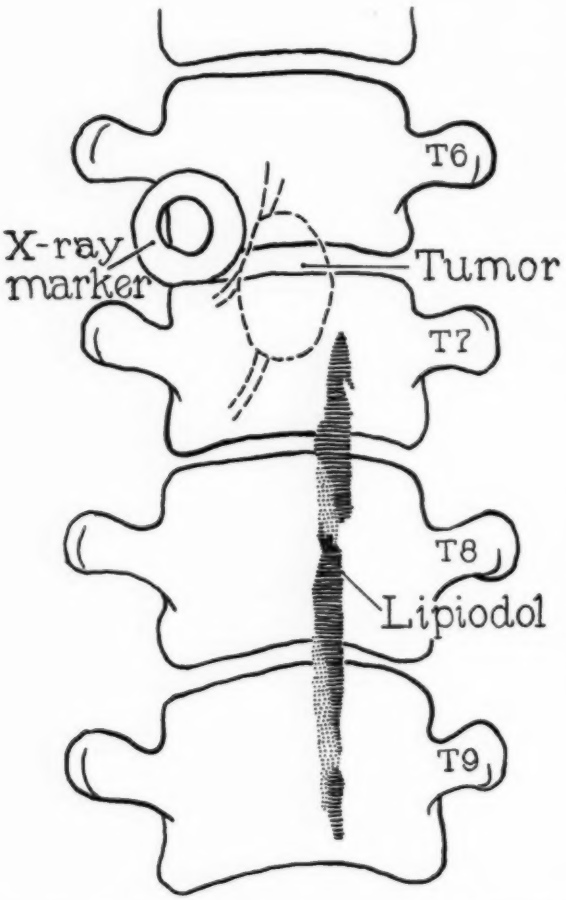


Fig. 15. Drawing from roentgenogram, Case III, showing arrest of campiodol at level of 7th thoracic vertebra.

History: The first symptom, which appeared eighteen months ago, was stiffness in the left knee. Almost simultaneously she lost control of both bladder and bowel sphincters. She states that she had been constipated for over two years. Sixteen months ago she complained first of pain in the territory of the left seventh thoracic dermatome. Twelve months ago for the first time she lost sensation to touch in the region of the thighs. Nine months ago she noticed weakness in the right leg. The paralysis has gradually increased until four weeks ago she was unable to walk even with assistance.

Neurological Status. Motor Disturbances: Spastic paralysis of both lower extremities.

Sensory Phenomena: Loss of pain, tactile and vibratory sensation to T. 7, loss of temperature sense in both lower extremities (see charts, Figs. 13 and 14). Pain was referred to an area on the left side of the abdomen 2 inches above the level of the umbilicus.

Reflexes: Patellar tendon reflexes exaggerated. Positive Babinski, right and left.

Cerebrospinal Fluid: Fluid xanthochromic. Protein, 21 units. Queckenstedt test, spinal block.

Roentgenogram: 1 c.c. of campiodol was injected into the lumbar sac, the patient's head lowered. The column of fluid was arrested at the level of the seventh thoracic vertebra (see Fig. 15).

Operation: October 14, 1931. Avertin anesthesia. Removal of laminae and spinous processes of T. 4, 5, 6, and 7. The tumor was situated on the posterior aspect of the cord (see Fig. 16). Two roots, probably the seventh and eighth thoracic, were injected with novocaine and silver clips applied. The tumor was removed together with the portion of the left posterior root, T. 8, which was attached to it. Wound closure. (Fig 17.)

Pathological Diagnosis: Perineurial fibroblastoma.

Postoperative Note: Immediate convalescence was uneventful. July 1932, the patient was able to walk with a cane, the incontinence had disappeared and sensation was partially restored.

Order of Appearance of Symptoms. An attempt was made to determine the chronological sequence of pain, paresthesia, and motor weakness. In 32 cases, or 82 per cent, pain was the first symptom; in 7 cases, or 18 per cent, paresthesias; and in 9 cases, or 23 per cent, motor weakness. Paresthesia and motor weakness occurred as the second symptom in 44 per cent and 26 per cent of cases, respectively. Pain as a second symptom was relatively infrequent. Motor weakness was found as a third symptom in 38 per cent of the cases, paresthesias in 13 per cent, and pain in 3 per cent. It seems to hold true, therefore, with extramedullary tumors that pain is overwhelmingly the first symptom noticed, and that paresthesia and motor

weakness appear later in the disease. Pain was present in all of the 39 cases, in 32 being the first symptom. Paresthesia was present in 33 cases, occurring chiefly as the second, sometimes as the first and third symptom. Motor weakness was found in all cases, chiefly as the second and third symptom. There are, however, exceptions, as in the following case:

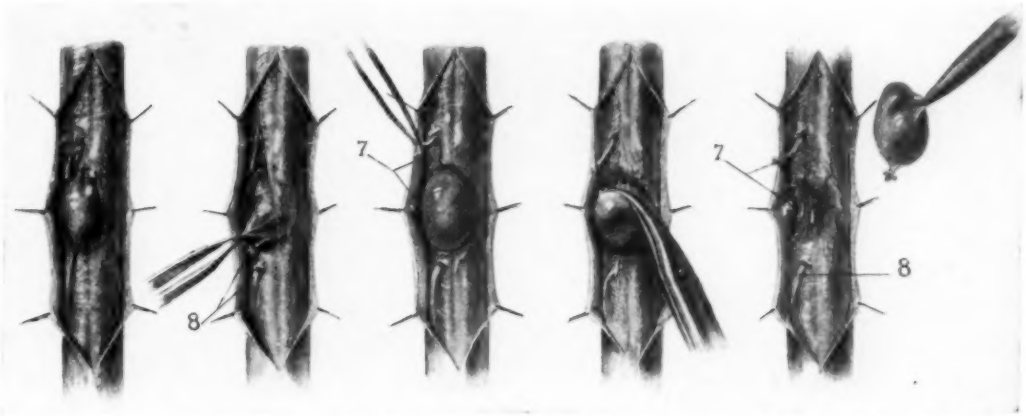


Fig. 16. Drawing at operation, Case III, showing tumor on the posterior aspect of the cord, involving the roots of the 7th and 8th thoracic segments.

Case IV. *Summary*. There are certain notable atypical features in this history although the physical characteristics of the tumor conform to the majority of our cases of fibroblastoma. The duration of the symptoms was only six months and pain did not precede the onset of motor weakness.

J. V. male, aged forty-two years (File No. 22577), was admitted to the Neurosurgical Service of the University Hospital September 2, 1931, complaining of weakness and stiffness in both legs and a pricking sensation in both legs and feet.

History: The patient was apparently in good health until six months ago, when he noticed that both legs became fatigued quickly on walking. At the same time he began to have a dull aching pain in the lower part of his back and right shoulder. The pain in his back remained localized to this area and did not radiate around the abdomen. The pain has remained constant in location and of the same intensity.

Eight weeks after the onset of weakness in his legs, he began to be troubled with a pricking sensation in the right foot This he described as "pins and needles." This disturbance of sensation gradually involved the entire length of the right limb and shortly afterwards the left limb. His legs have become progressively weaker so that his gait is slow and faltering, and at times they feel so heavy he can hardly lift them. Often he complains of coldness in feet and legs. For the past two weeks there has been difficulty in urination.

Neurological Status. Motor Phenomena: The patient cannot stand on his right foot, and he walks with a dragging uncertain gait. The loss of power in the right leg is quite marked but only slight in the left leg. The dynamometer registers 90 in the right, and 70 in the left hand.

Reflexes: All abdominal reflexes, upper and lower, and the cremasteric are absent on both sides. Biceps and ulnar reflexes are normal and equal. Triceps reflex is slightly diminished, equally on both sides. No ankle clonus or patellar clonus; positive bilateral Babinski.

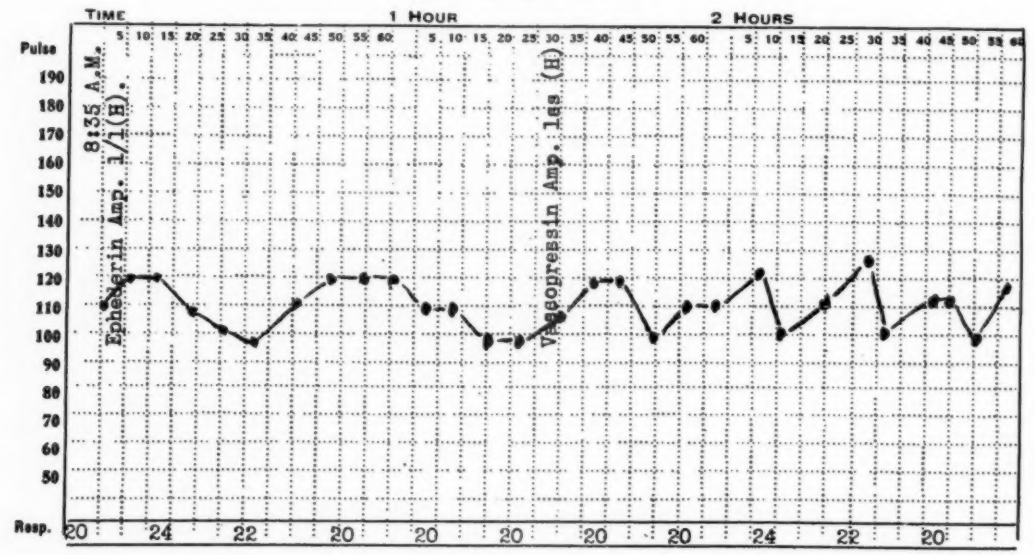
Sensory Phenomena: Pain sense is lost on the left side up to the fourth interspace in front and up to the level of the sixth spinous process behind. On the right side pain sensation is diminished up to the crest of the ilium front and back, and from this level upwards pain sensation is lost up to the fourth interspace. Temperature sense is lost up to the fifth thoracic dermatome, and from the fifth to the third dermatome diminished, with certain exceptions (see charts, Figs. 18 and 19). Complete loss of tactile sensation is limited on the left side to an area supplied by the second lumbar nerve and on the right side by the first lumbar nerve. Tactile sensation is diminished

FORM A-11

ANESTHESIA AND BLOOD PRESSURE RECORD.

NAME | HOSPITAL | INDEX FILE NO. | VOL.

ANESTHESIA CHART.



Operation Laminectomy with removal of cord tumor

Date 10.14.31

Operator Dr. Frazier

Operation Started 8.40 A.M.

Operation Ended 11.30 A.M.

Hypodermic Medication (Codein gr. ss. (Atropine gr. 1/150

Anesthetic Avertin, local

Variety

Method Rectal

Anesthetizer Miss Hitz

Time to Anesthetize 30 minutes

Amt. to Anesthetize avertin 6 c.c. distilled water 240c.c.

Total Amt. used avertin 6 c.c. Distilled water 240 c.c.

Examination of chest (before)

BLOOD PRESSURE CHART.

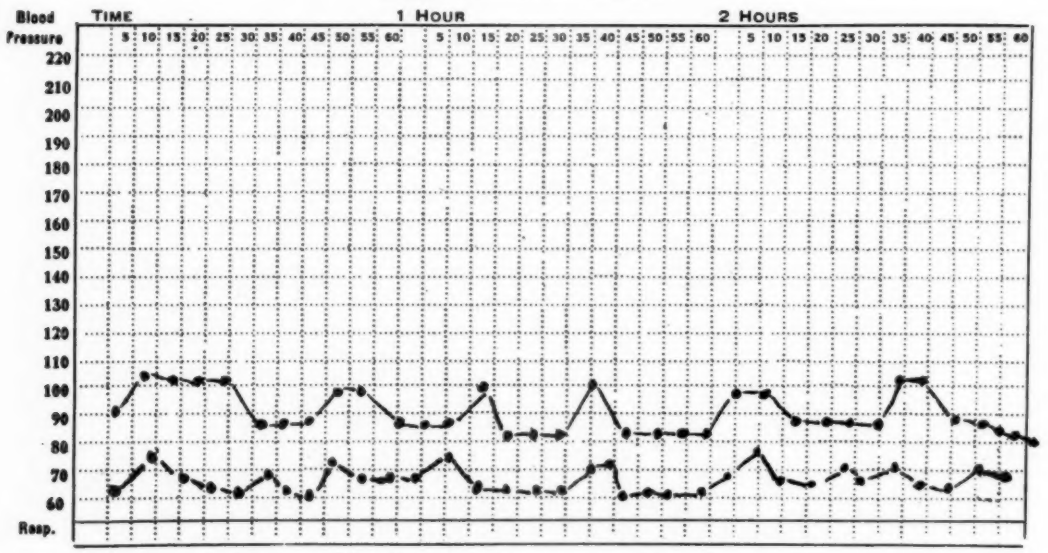


FIG. 17. Blood pressure chart at operation, Case III.

to the tenth thoracic dermatome on the right side.

Cerebrospinal Fluid: Pressure 73 mm., rising to 310 mm. upon jugular compression, falling to 165 mm. in two minutes and to 152 mm. upon withdrawal of 10 c.c. of fluid. Upon coughing the pressure rose to 340 mm. from 150 mm., and upon straining to 185 mm. Globulin 9 units; lymphocytes from 2 to 3, colloidal gold test 0112432100. Wassermann negative.

Operation: Laminectomy, September 15, 1931. Avertin anesthesia, supplemented with ether. Removal of spinous processes and laminae of T. 1, 2, 3, 4. An intradural tumor occupying the anterior aspect of the second and third thoracic segments was found and removed (see Fig. 20).

Postoperative Note: After an uneventful recovery from the operation the patient was discharged September 30, 1931. A report received on August 24, 1932 states that the patient walks very well with a cane. No trouble with sphincter control, power in legs slightly diminished but no sensory loss in legs. Reflexes in lower extremities still slightly hyperactive on both sides. The patient is well satisfied with the results.

Comment: The duration of the symptoms in this case was only six months, a considerably shorter period than the average, about thirty months. Although pain usually precedes motor weakness, both appeared in this case at the same time, but it must be remembered that the tumor was so situated as to press directly upon the pyramidal tracts. The pain in the lower part

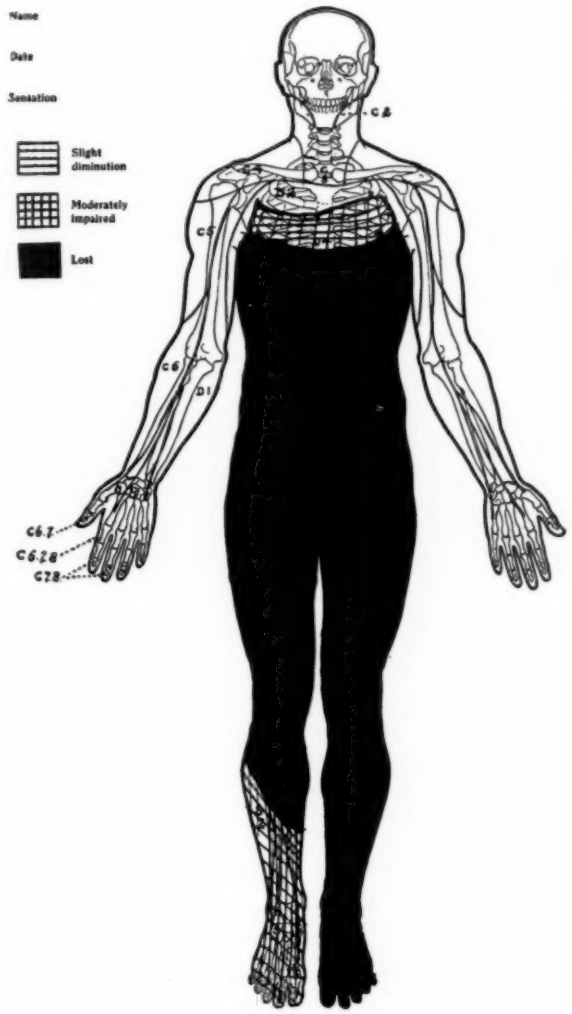


Fig. 18.

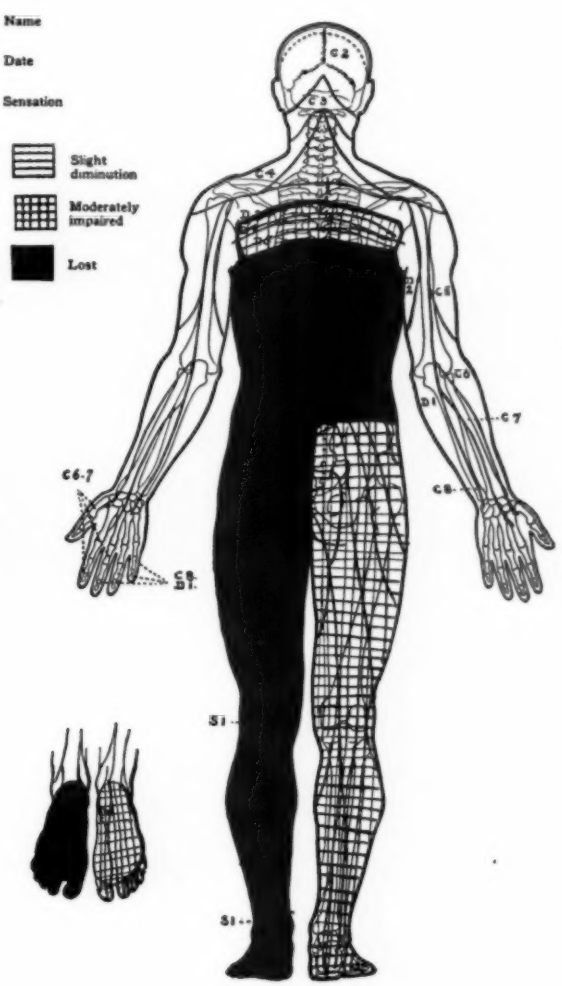


Fig. 19.

Figs. 18 and 19. Sensory charts, Case IV, showing loss of temperature sense.

of the back was misleading as a localizing symptom, the tumor being at the level of the second and third thoracic segments, while the pain in the shoulder suggested a higher level. The motor weakness and paresthesia, which began below and extended centralwards, became more pronounced as the pressure increased.

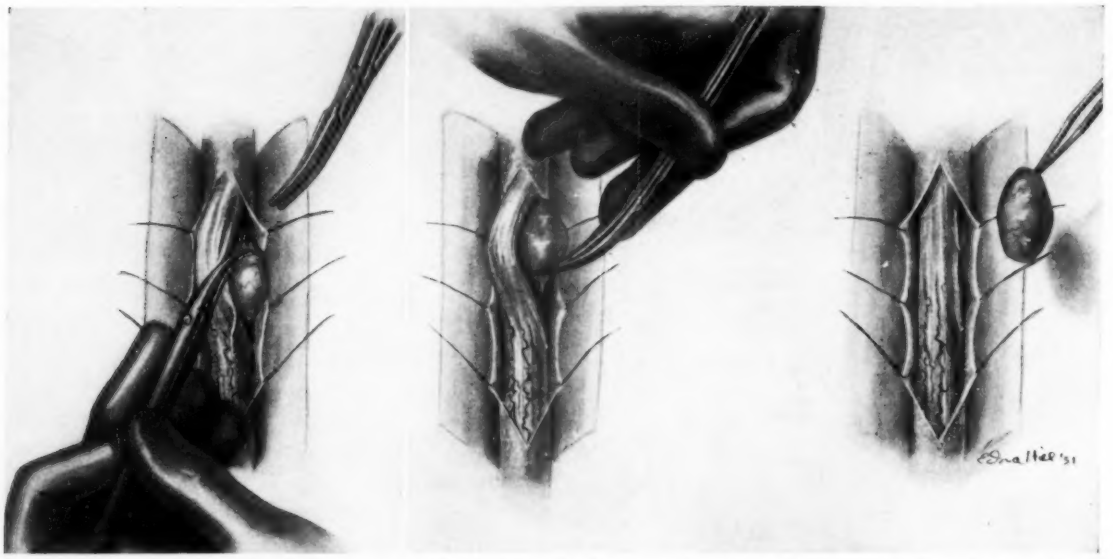


Fig. 20. Drawing at operation, Case iv, showing removal of tumor from anterior aspect of cord at 2nd and 3rd thoracic segments.

Some time ago Frazier[8] divided the life history of spinal cord tumors into three cycles. The following table (Table iv) confirms this contention. The first, the root or pain cycle, is usually the longest. That pain should be an initial symptom so frequently is readily understood when one recalls the origin of the extramedullary tumor from the reflection of the arachnoid over the root at its point of exit. (Fig. 21.)

The early onset of pain and its long duration before other signs of tumor appear readily explain the number of mistakes that have been made in diagnosis. It has been estimated that these mistakes have been made in 10 per cent of any series, and in many of these cases pain has been attributed to disease of the gall bladder, stomach, duodenum, appendix or pelvic structures. Neuritis, arthritis, tuberculous spondylitis are among other diseases for which the patient may have been treated.

Consider now the position of the tumor in relation to the appearance of pain, paresthesia and motor weakness (see Table ii). Pain is a prevailing symptom of extramedullary tumors, no matter on what surface of the cord the tumor may be. It was most frequently present when the tumor was on the lateral aspect of the cord, either directly lateral, anterolateral or posterolateral; but only occasionally when the tumor was on the anterior aspect. Weakness did not appear early in every case of anteriorly situated tumors, but when present, the paralysis was usually advanced. Paresthesias were constant symptoms with this exception; they were notably absent in tumors on the lateral aspect of the cord.

It is interesting to note the chronology of the symptom sequence according to the relation of the tumor to the meninges; that is, whether extradural or intradural. In 27 of the 39 cases the tumor was intradural, in 5 cases extradural, and in 6 cases they were described as both extradural and intradural. In 27 intradural tumors pain was the first symptom in 22, or 81 per cent, and a rather prominent feature in the others. Paresthesia was prominent as a

second symptom in 16 cases, or 59 per cent, and weakness was the third symptom in 44 per cent. In 33 per cent of the intradural tumors, weakness was the first symptom and in 15 per cent a second symptom.

TABLE IV
ORDER OF APPEARANCE OF PAIN, PARESTHESIA AND MOTOR WEAKNESS

Order of Appearance	*Pain*		*Paresthesia*		*Motor Weakness*	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
1st	32	82.0	7	18.0	9	23.1
2nd	3	7.6	17	43.6	10	25.7
3rd	1	2.6	5	12.8	15	38.4
4th	1	2.6	2	5.1	3	7.7
5th	1	2.6	2	5.6	2	5.1
6th	1	2.6	0	0	0	0
Absent	0	0	6	15.4	0	0

Of the 5 extradural tumors, pain was the first symptom noted in all cases. Weakness appeared first in 2 instances, was the second symptom in one case, the third in one case, and the fifth in the remaining case. Paresthesia appeared as the first symptom in 3 cases; the third symptom in one, and the fifth symptom in the remaining case.

In 6 cases of tumors both intradural and extradural, pain was the first symptom noted in 3, paresthesia in 3, and weakness in one. Paresthesia appeared once as a third symptom and in the remaining case was absent. Weakness was the first symptom in one case, the second symptom in 4 cases, and absent in one case.

As to the course and duration of symptoms, the average duration of the symptoms before the patient was admitted to the Neurosurgical Clinic was 29.1 months for fibroblastoma, and 8.5 months for fibroma.

The shortest period was five months and the longest seven years. Occasionally symptoms may appear with surprising suddenness. In some instances a complete paraplegia may develop in two days. It is generally believed that patients may survive tumors of the cauda equina for a

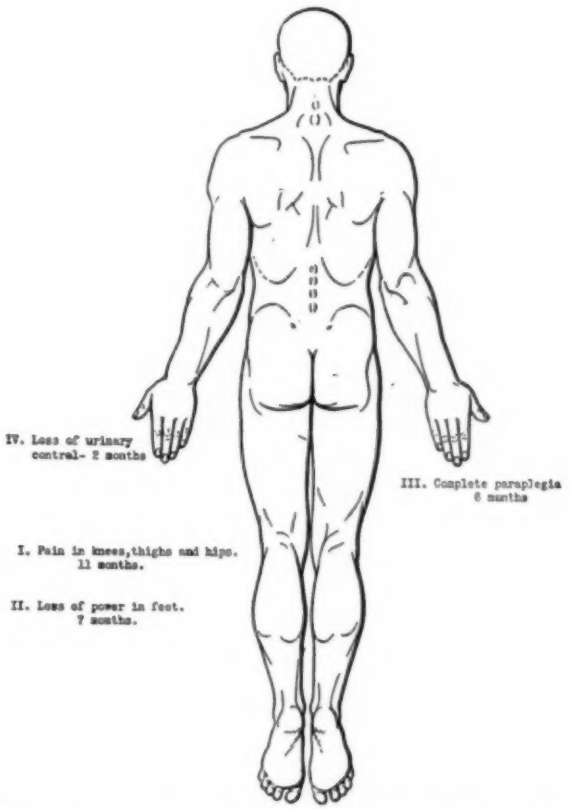

IV. Loss of urinary control- 2 months
III. Complete paraplegia 6 months
I. Pain in knees, thighs and hips. 11 months.
II. Loss of power in feet. 7 months.

FIG. 21. Composite chart showing chronological order of appearance of symptoms.

longer time than in other sections of the cord. This may be due to relatively greater space in that part of the vertebral canal.

A comparison of the duration of the various symptoms is interesting, especially that of the duration of pain, of paresthesia and of motor weakness. In Table v one notices a considerable lapse of time between the appearance of pain and that of paresthesia or motor weakness. For instance, with the fibroblastoma, the average duration of pain was 27.7 months, of paresthesia 17.0 months, of motor weakness 16.5 months. The interval between the appearance of paresthesia and of motor weakness was very short.

Periods of remission have frequently been observed in this series. In one case, the patient was free from pain for six years and at the end of this period the pain re-

appeared. In another case, stiffness of the great toe disappeared a short time after its onset, to recur after an interval of several months. In some instances there was a definite transitory improvement when the symptoms recurred with increased severity. Sometimes there may be a change in the picture, occasionally without apparent cause, or more often after lumbar puncture. In only 2 cases of this series did a sudden aggravation of symptoms follow lumbar puncture.

TABLE V
DURATION OF SYMPTOMS BEFORE OPERATION IN MONTHS

Type of Tumor	*Total*	*Pain*	*Paresthesia*	*Motor Weakness*
Fibroblastoma....	29.1	27.7	17.0	16.5
Fibroma.....	8.5	5.6	5.7	6.2
Neuroma.....	5.0	5.0	5.0	5.0

LEVEL DIAGNOSIS

When a spinal cord tumor suspect appears in the Clinic the neurosurgeon has two responsibilities: to eliminate other lesions, such, for example, as transverse myelitis, syringomyelia or multiple sclerosis, and, having eliminated these, to determine the level of the lesion. The differential diagnosis between neoplastic and non-neoplastic lesions is not always easy. A number of mistakes have been made in this Clinic and usually for two reasons: either the history was atypical or there was no definite, unmistakable level of sensory loss, whether partial or complete. By atypical histories are meant those in which pain either plays no part at all or a very inconspicuous part. Syringomyelia should be readily recognized, but some of the degenerative processes may lead one into errors of diagnosis. Tumors should not be confused with cases of transverse myelitis. The course of the latter is more rapid, the cell count is increased, there may be a history of lues or positive serological reactions, the motor overshadows the sensory picture. Yet mistakes are made and were made in a number of cases as the one below cited. Despite the fact that the patient was the mother of three healthy children, with a negative serology and with no increase in cells, she had been branded on several occasions prior to her admission to our Clinic as a case of transverse myelitis and denied the benefits of operation. In the absence of any suggestion of lues, when there is a definite level of disturbed sensation, when paralysis is progressive, when the level diagnosis can be established without any reasonable doubt, that patient is entitled to the possible benefits of an exploratory operation. And so in this case, though the diagnosis was not clearly established, an exploratory operation was advised, a tumor was found and removed.

CASE V. *Summary.* A large extradural fibroblastoma was found in a patient whose history had been interpreted in various clinics as one of transverse myelitis. Although the tumor presented on the posterior aspect of the cord, the first symptom was motor weakness, preceding pain by two years. The tumor was successfully removed and the patient made a complete recovery.

Mrs. A. J. H., aged thirty-seven years (File No. 63196), was admitted to the Neurosurgical Service of the University Hospital December 4, 1920, with the following history. She was well until the summer of 1917 when she noticed that the right great toe was stiff. She was aware of it only when she attempted to rise on the toes in tennis or golf. She remembers no pain associated with the stiffness. This stiffness disappeared after a short time. In December, 1917 she noticed that she could not balance properly when she tried to skate. During the early months of 1918 there was the same difficulty in skeeing. There was no difficulty in the ordinary movements of walking. In the fall of 1919 there appeared a stiffness in the thighs. In dancing she could not move with her usual spring. About this time there were occasional cramp-like pains in the feet which came on during exertion, relieved by taking off the shoes. She could walk without difficulty while on level ground, but going up inclines and stairs was difficult. During all of this there was no pain in the legs. She began to use a cane August, 1919, and by the spring of 1920 voluntary movement

of toes and ankles, knees and hips was lost. There has been some tingling in both legs and during the early part of her illness there was

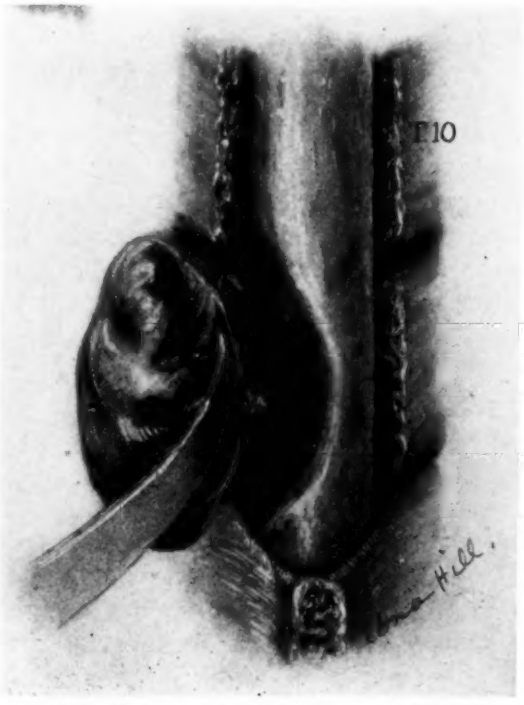


Fig. 22. Drawing at operation, Case v, showing tumor with mushroom-like stalk at level of laminae of T. 10 to T. 11.

hypersensitiveness in the soles of the feet. She has no difficulty in appreciating temperature.

Cerebrospinal Fluid: Pressure 78 mm. Fluid clear and colorless. Cells 8 per c.cm. Globulin trace. Wassermann and colloidal gold negative.

Neurological Status (Notes by Dr. Spiller): There is considerable spasticity of each lower limb, more of the right. There is some voluntary movement of the toes, of both ankles and of the knees and hips. But all voluntary movement is very slight.

Reflexes: The patellar reflex is distinctly exaggerated on each side, and equally, and patellar clonus is persistent on the right and indicated on the left. Persistent ankle clonus on each side and about equal on the two sides. Babinski reflex typical on each side.

Sensory Phenomena: Heat and cold sensations are greatly diminished in all parts of the lower limbs, almost to Poupart's ligament on each side. But these sensations are entirely normal in the abdomen. The disturbance of these sensations includes the first lumbar segment but not the twelfth thoracic. Pin prick apparently is nearly normal in the lower extremities. Tactile sensation seems to be the same as heat and cold. Sense of position is lost

Fig. 23. Tumor, Case v, after removal intact. Fibroblastoma.

in each big toe. In the perianal region of each side there is slight impairment of touch, apparently none of pain. There is considerable impairment in the perianal region to heat and cold.

There is perfect control over bladder and bowels.

There is no deformity of the vertebral column and no tenderness over the spine.

The symptoms are those of an incomplete lesion of the spinal cord about the first lumbar or twelfth thoracic segment. The preservation of control of the bowels and bladder is indicative of the incompleteness of the lesion. There has been burning pain in the feet in the early stage of the lesion and not only during exertion, as she has had it in bed. The symptom complex is not unlike that of an anemia but the blood examination does not show an anemia. If it were from pressure on the cord, one would expect greater spasticity. The indications are rather that of an intramedullary lesion or a partial transverse myelitis. Tumor is possible but seems doubtful. Voluntary movement in the lower limbs has been increasing.

Operation: Laminectomy December 14, 1920. Exposure and removal of an extradural tumor. On the assumption that the tumor to be at the level of the first lumbar segment, it was planned to remove the spinous processes and laminae of the 8th, 9th, and 10th thoracic

vertebra. This was done beginning above and going down and when the 10th lamina was removed there appeared, projecting from below, a bluish colored tumor which seemed to occupy approximately three-fourths of the spinal canal, the cord being displaced well over to the right side. The tumor began at the lower margin of the laminae of T. 10 and extended down for a distance of 6 cm. necessitating removal of the laminae and spinous processes of T. 11. This enabled us to remove the tumor intact. The shape of the tumor was rather peculiar in that there projected to the left and forwards a tongue-like process like the stalk of a mushroom. (See Figs. 22 and 23.) So far as could be seen it was entirely encapsulated and the tumor was removed intact. Two bleeding points were controlled with muscle grafts.

Postoperative Notes: There was an extraordinarily rapid recovery of function. Three weeks after the operation the patient could walk and sensation was entirely normal with the exception of an area of hyperesthesia over the left ankle and foot. Today, twelve years after the operation the patient is wholly symptom free.

SEGMENTAL DIAGNOSIS

To determine the segmental level we rely upon (1) sensory phenomena, (2) vasomotor disturbances, (3) sympathetic phenomena, (4) motor symptoms such as atrophies or twitchings, (5) loss of an individual reflex (see Case VI), (6) injections of iodized oil, and (7) dual lumbar punctures.

Cervical Region. Of the 12 tumors in the cervical region, there was a definite sensory level in all but 2. In 5 cases the sensory level corresponded with the level of the lesion, and in one case it was one segment lower than the tumor level. In 2 cases there was a difference of three or four segments between the level of anesthesia and the level of the tumor. In one of these, a tumor of large dimensions, the sensory loss was three segments higher than the tumor; in another the tumor was found at C. 2 and the highest level of disturbed tactile sensation corresponded to C. 6. In another case with a sensory level corresponding to the third cervical segment, the tumor was located on the posterolateral aspect of the cord compressing the fifth, sixth and seventh cervical segments and affecting the corresponding posterior roots.

Sympathetic phenomena are more frequent with tumors in the lower cervical and upper thoracic regions. In 5 of the 12 cases of cervical tumors there were sympathetic disturbances. In 2 cases of tumor, extending from the medulla oblongata to C. 1 and 2, the pupil was dilated and in one there was an acceleration of pulse. In 3 cases the pupil was contracted and in one case there was an enophthalmos.

Atrophy and twitching were observed in 8 cases; in 2 of these the tumor extended from the lower part of the medulla oblongata to C. 1 and 2. In one of these there was bilateral atrophy of the tongue and atrophy of the muscles of the shoulder and arm; in the other there were involuntary contractions of the trapezius. As the phrenic nerve derives its innervation from the third, fourth and fifth cervical segments, a tumor compressing these segments may cause paralysis of the diaphragm. But of 7 tumors affecting these segments, in only 2 was the diaphragm involved. Atrophy of the peripheral muscles of the hand was seen not only in the lower but also in the upper cervical tumors. Fibrillary twitchings of the deltoid, triceps and biceps were observed with a tumor at the fourth cervical segment.

Generally, the reflexes of both upper and lower extremities were exaggerated, whereas the abdominal reflexes were diminished or absent. There were 2 cases of tumor at the seventh cervical segment, in one of these the triceps and the biceps reflexes were normal, in one the triceps reflex on one side was lost; and in one case of tumor of the fourth cervical segment, as the following, the biceps reflex was lost on one side and exaggerated on the other.

Case VI. *Summary.* The early knife-like pain, group muscle atrophy and tremors, weakness of one side of the diaphragm, the loss of the right biceps tendon reflex and exaggeration of the left, and preservation of power in the lower extremities were the characteristic features in this history. The patient made a complete recovery after the removal of a subdural fibroma.

T. S., male, aged twenty-nine years (File No. 65039½), was admitted to the Neurosurgical Service of the University Hospital, February 17, 1921, complaining of pain in the right shoulder.

History: September, 1920, while engaged in heavy work, he experienced a knife-like sensation on the right side of the neck, radiating to the shoulder. The pain was aggravated by movements of the arm or neck and by jarring. In January, 1921 the pain was so severe he was forced to give up his work, and since then, because of a painful throbbing in the right shoulder, he has not been able to sleep comfortably.

In February, 1921, his ankles suddenly became swollen and he developed pain in both legs from his feet to a few inches above his knees. Later the pain in the neck extended to the elbow, forearm and hand. The right hand was numb as well as painful and not fully under control.

Neurological Status: Motor Phenomena: Intense atrophy of the infraspinatus and supraspinatus muscles, right, and to a less degree of the muscles of the shoulder girdle. Marked fibrillary tremors of triceps, biceps and deltoid muscles, right. Restriction of movement of left side of diaphragm.

Sensory Phenomena: Hyperalgesia in the distribution of the right third and fourth cervical nerves. Pain and temperature sensation impaired in the left lower limb and left side of trunk. Tactile sensation, not disturbed.

Reflexes: The right biceps reflex is absent. Tapping the biceps tendon caused only pronation of the forearm. Left tendon biceps reflex is normal. Right and left triceps tendon reflexes exaggerated, the right more than the left. Patellar and tendon reflexes equally exaggerated. Bilateral patellar clonus, ankle clonus right and questionable Babinski, right.

Cerebrospinal Fluid: Clear. Pressure 52 mm. Cell count, 6. Globulin and Wassermann negative.

Operation: Laminectomy, June 4, 1921. The tumor was found as anticipated on the right side of the cord, about the level of the fourth

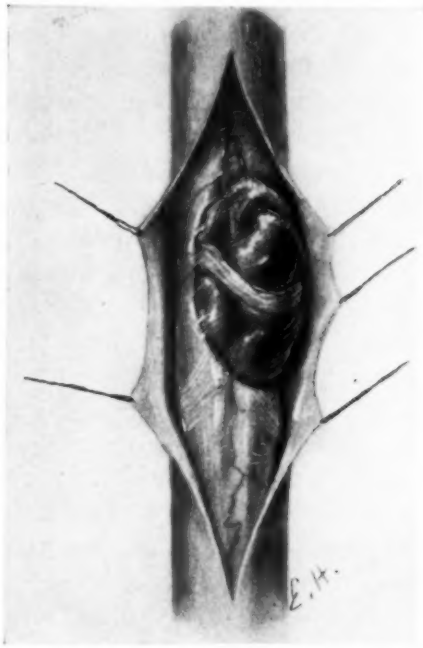


Fig. 24. Drawing at operation, Case VI, showing fibroblastoma at level of 4th cervical segment.

cervical segment (see Fig. 24). The tumor, which extended 1 cm. into the intervertebral foramen, was removed *in toto.* The patient was discharged June 26, 1921, after an uneventful convalescence.

Pathological Diagnosis: Fibroma.

Postoperative Note: When last seen six years after operation there had been no sign of recurrence.

Thoracic Region. Definite sensory levels were found in all 23 cases of tumors of the thoracic region. In 15 cases, or 65 per cent, they corresponded precisely and in 6 cases, or 26 per cent, to within one segment of the level of the lesion. In 2 cases the anesthetic level was two segments lower, and in one three segments lower than the tumor. Of the different forms of disturbed sensation, pain, temperature and touch were the most constant. In 4 cases sensibility for pain and temperature was disturbed and tactile sensibility preserved.

Sympathetic symptoms were observed in 4 thoracic tumors. In one case, a lesion at

the eighth cervical and first and second thoracic segments, there was a contracted pupil and a narrow palpebral fissure. In 2 cases in which the vasomotor and pilomotor level was determined both corresponded with the level of sensory loss.

Motor phenomena, such as twitching and atrophy, were of little assistance in determining the segmental level of thoracic tumors. In a tumor on the anterolateral aspect of the third and fourth thoracic segments there was atrophy of the left deltoid muscle and of the muscles of the left hand. Pain, temperature and touch sense were lost up to the fourth interspace.

In all of the thoracic tumors the reflexes of the lower extremities were increased. Increase of tendon reflexes was usually accompanied by pathological reflexes, such as ankle clonus, patellar clonus, a positive Babinski, Gordon, etc. and reflexes of defense were observed in many cases. As with cervical tumors, the abdominal and cremasteric reflexes were usually absent.

Lumbar Region. There were but 3 tumors of the lumbar segments, all with sensory levels; 2 corresponded to the tumor level; in one the sensory level was four segments higher. In one of the three, tactile sensation was preserved; in the other two, pain, temperature and tactile sensations were disturbed. A vasomotor level was observed in one case which appeared to be four segments higher than the tumor level. In none of the lumbar series was there muscle atrophy or twitching. The patellar reflexes were exaggerated in all 3 cases and in one, where the tumor extended from the twelfth thoracic segment to the tip of the conus, the lower abdominal reflex was recorded as diminished.

Caudal Tumors. One tumor was found in the cauda equina. There was no sensory level. According to Spiller[25] the development of symptoms in cauda equina lesions usually is very gradual. The onset is characterized by pain and loss of reflexes in the lower limbs, followed often much later by paralysis, atrophy and anesthesia. Pain occurred very early in the case in this series, the Achilles tendon reflex was lost, the patellar tendon reflex diminished, and the paralysis was flaccid.

CEREBROSPINAL FLUID

Examination of the cerebrospinal fluid often gives valuable information. Xanthrochromia, increased globulin content without increase of cells, a complete or partial block are the characteristic findings. A sudden change in the course of the disease, after lumbar puncture, has been reported frequently. In one of our series the patient, though able to walk before lumbar puncture, lost all power in the lower extremities afterwards. In another instance there was a marked aggravation of both motor and sensory symptoms after lumbar puncture. Davidoff[26] has reported a similar experience.

The presence of xanthrochromia in tumors of the spinal cord depends upon various factors; the level of the lesion, how complete the block, and the duration of the block. In tumors of the cervical region xanthrochromia is not so frequent as in the lower segments, and when present the yellow discoloration is not so intense. According to Stookey,[27] the darker shades of yellow occur below the fifth thoracic segment, and the lighter shades above this level. Elsberg and Constable[28] believe that deep orange-yellow fluids are met with almost exclusively in the lower levels of the cord. Thirteen of the 39 tumors of this series, or 33 per cent, showed xanthrochromia, including only 2 cervical tumors.

The globulin content was recorded in 25 cases and in 15 cases, or 60 per cent, it was definitely increased. The increase of globulin does not seem to depend upon the level of the tumor. The number of cells of the cerebrospinal fluid was recorded in 27 cases, and in none was there an increase in the cell content.

SUMMARY AND CONCLUSIONS

1. Thirty-nine cases of extramedullary tumors of which thirty-four were fibroblastomas are studied and analyzed.

2. The clinical picture is reviewed (1) from the standpoint of the chronological appearance of the symptoms and (2) from the position of the tumor with relation to the circumference of the cord.

3. Sensory and motor phenomena are discussed, together with disturbances of sphincters with respect to diagnosis and the location of the tumor.

4. Pain was found constantly and early, regardless of the level of the tumor. It was the initial symptom in 83 per cent of all cases.

5. Next to pain paresthesia was the most constant symptom. It was present in 80 per cent of the cervical, 55 per cent of the thoracic and in all lumbar and caudal tumors. Paresthesias may be homolateral, contralateral or bilateral.

6. Paralysis was spastic in all but the one case of caudal tumor.

7. For level diagnosis the level of sensory loss is the most informing. Of the different types of sensation, the loss of pain and temperature sense was the most constant.

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CRANIOCEREBRAL INJURIES*

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THE subject of head injuries has received much attention in the current medical literature, due partly to the fact that the incidence of skull fracture is definitely on the increase, and partly to the fact that there still exists much confusion in regard to the management of these cases. Mock recently reviewed over 20,000 cases of head injury occurring since the beginning of this century. This author draws attention to the fact that the treatment in each decade varied considerably; for example, during the period from 1900 to 1910, the treatment was conservative; from 1910 to 1920 decompression operations became the vogue with an unusually high mortality. This operative era was again followed, during the years from 1920 to 1928, by an era of conservatism, the treatment consisting essentially of prolonged rest in bed and supportive measures. During the past few years, newer concepts regarding the care of skull fractures have been established, the work of Temple Fay being particularly outstanding. Fay's contribution to this subject has been the establishment of the so-called dehydration therapy upon sound physiological principles.

It is the purpose of this paper to compare the results of two separate series of cases of craniocerebral injury, one of which was treated by expectant or supportive measures, and the other by dehydration methods. The first series, 54 in number, comprised all cases which were admitted to the Denver General Hospital during the first eight months of 1927, while the second series, 60 in number, were those cases admitted during the first eight months of 1932.

In both series of cases the history of the injury was essentially the same, the predominating cause of injury being automobile accidents, accounting for 72 per cent and 78 per cent of all the skull fractures during the first eight months of the years 1927 and 1932 respectively. (Table 1.) This is not surprising in view of the fact that Denver is not an industrial or manufacturing center. Furthermore, the number of accidents tabulated here probably represent more than 95 per cent of the total number of skull fractures seen in Denver, inasmuch as the Denver General Hospital is the only receiving hospital in the city.

TABLE 1

	1927		1932	
No history obtainable	1		2	
Assault	3		3	
Automobile collision	14	72%	19	78%
Struck by automobile	19		23	
Fall from moving vehicle	6		5	
Fall from a height	10		7	
Gunshot	1		1	

The physical findings in the reported cases were similar. Lacerations or contusions of the head were present in 85 per cent of the cases in 1927, and in 80 per cent of the cases in 1932. Bleeding from one or both ears was a complication in 30 per cent of the cases in each series, while ecchymosis of the eyes occurred in 20 per cent and in 26 per cent of the cases in 1927 and 1932 respectively. Fractures in other bones throughout the body were found in 25 per cent of those patients presenting themselves in 1932, while this

* From the Department of Surgery, Denver General Hospital. Presented before the Midwinter Post-Graduate Clinics, Denver, January 18, 1933.

complication was only present in 13 per cent of the 1927 series. A rather marked difference, however, existed in the degree of unconsciousness, indicating perhaps the more serious nature of the injuries occurring in 1932. A state of unconsciousness on entry to the hospital, or a definite history of unconsciousness occurred in 52 per cent of the cases in 1927 and in 60 per cent of the cases in 1932. In the former instance, the length of time that unconsciousness persisted varied from a few minutes to forty-eight hours, the average period of unconsciousness in 27 cases being 6.3 hours. In the latter instance, the unconscious period varied from a few minutes to eleven days, the average period of unconsciousness in 33 cases being 18.7 hours. Briefly then, unconsciousness was present in 8 per cent more patients in 1932 than 1927, and the period of unconsciousness in the 1932 series was three times longer than in 1927. Other associated findings, such as internal injuries, alcoholism, drug addiction, epilepsy, lues, arteriosclerosis, etc., occurred in a few isolated instances in each series of cases.

MORTALITY

Mortality statistics, as reported by various authors, are found to differ rather widely, depending upon the types of cases included in the report. For example, a comparatively low mortality rate may result where all types of cerebral trauma, including cases showing or giving a history of momentary unconsciousness, are included. Frequently, also, patients dying upon or shortly after entry to the Emergency Room are not included due to the fact that they have not been officially admitted to the hospital. In order to avoid misunderstanding, only those cases were chosen for this report which showed definite evidence of a skull fracture, as proved by *x*-ray examination, or laceration of the brain, as shown by blood in the spinal fluid, or both. In a certain few instances, death supervened so rapidly that lumbar puncture or *x*-ray was obviously not attempted, but the diagnosis was established in these cases by the autopsy findings.

The mortality was 31.5 per cent in the series of 1927 and 28.3 per cent in 1932, a difference of 3.2 per cent. (Table II.)

Table II
MORTALITY STATISTICS

	1927	1932
Number of cases	54	60
Number of deaths	17	17
Mortality (per cent)	31.5	28.3

Of those individuals dying in 1927, the shortest survival period was twenty minutes, and the longest was seventy-two hours, the average length of time in hours before death supervened being 17.4 hours. In 1932, the shortest survival period was thirty minutes, and the longest was thirty-eight days, the average time in hours being 84.8. However, exclusive of this one patient surviving thirty-eight days, the average survival period in 1932 was 33.1 hours, or almost twice that of 1927. The majority of the patients died in the first twelve hours, as may be seen from an examination of Table III. Obviously, at least 50 per cent of the cases reported in Table III may be classed as so-called inevitable fatalities, inasmuch as the patients succumbed so quickly that shock measures only could be instituted, without regard to the treatment of the craniocerebral injuries.

Table III

Deaths	1927	1932
In less than 1 hour	1	2
In less than 2 hours	3	1
In less than 3 hours	3	1
In less than 4 hours	1	0
In less than 12 hours	4	6
Total in less than 12 hours	12 or 70%	10 or 60%

In the reported deaths for 1927, associated or complicating injuries of a serious nature were described in only two instances: in one case a fractured humerus, and in the other, fracture of several ribs. From the subsequent course in these cases, it did not appear that these injuries greatly affected the outcome. On the other hand, in those deaths occurring in 1932, there were three patients who in addition to sustaining skull fractures, also received other injuries which were of a sufficient magnitude to have been of themselves the primary cause of death. The first case was that of a man eighty years of age, who, in addition to a small linear skull fracture, suffered a compound fracture of the femur. The second case was that of a young man of thirty years, who on admission to the hospital was found to have a fracture of both femurs and the left ulna. Within three days he developed meningitis and at autopsy a fracture of the frontal bone was discovered. The third case was that of a girl of twenty years who died thirty minutes after entry to the hospital. Post-mortem examination revealed rupture of the liver and right kidney in addition to a skull fracture. Therefore, if these 3 cases were excluded, the mortality for 1932 would be approximately 24.6 per cent as compared to the mortality of 31.5 per cent occurring in 1927, or a difference of 6.9 per cent.

TREATMENT

Obviously the preliminary shock which is present in the vast majority of head injuries should be given the first consideration. Perhaps it may seem unnecessary to mention this frequently repeated statement, but it appears from reviewing these cases that this fact has been to a certain extent overlooked in the past. Detailed physical examinations during the period of shock should be postponed, time should not be wasted in the repair of extensive lacerations, nor should early *x*-ray pictures be taken.

In both series of cases, the preliminary shock was treated by the ordinarily acceptible methods, with the exception that intravenous therapy was used much more extensively in 1932 than in 1927. Furthermore, a review of these cases disclosed the fact that in 1927 roentgenograms were frequently obtained during the period of shock. The time after entrance to the hospital until *x*-rays were taken could be determined in 35 of these cases, the average time being 9.4 hours. However, in 16 cases, or nearly 50 per cent, *x*-rays were taken within one and one-half hours after admission. On the other hand, in 1932, *x*-ray examinations were not permitted until all evidences of shock were past, usually after a delay of several days.

Following the period of shock, we must concern ourselves with the cerebral injury itself. The treatment of those cases occurring in 1927 may be summarized with the statement that no specific measures were instituted, and the therapy consisted essentially of supportive measures and rest in bed, with frequent diagnostic lumbar punctures and the occasional use of small doses of magnesium sulphate.

The treatment in 1932, on the other hand, has been directed toward the reduction of the cerebral edema and increased intracranial pressure by dehydration means.

In these cases, the intracranial pressure was reduced in the following four ways: (1) by repeated spinal punctures; (2) by the use of intravenous glucose solution; (3) by the limitation of fluids; and (4) by the use of magnesium sulphate, given orally or by rectum.

Spinal puncture as a routine measure is condemned by some authorities on the basis that sudden death may supervene from descent of the medulla into the foramen magnum. We believe, however, that it is the consensus of opinion that this danger is exaggerated, and there can be no question of the advantage of this maneuver. An early diagnostic lumbar puncture was done in this series of cases

to determine the character of the spinal fluid and the degree of intracranial pressure. In the presence of increased pressure, an amount of fluid was removed sufficient to reduce the spinal fluid pressure by more than 50 per cent, and subsequent spinal punctures were repeated at eight to twelve hour intervals, being guided by the blood-pressure findings, the pulse rate, and each subsequent spinal pressure reading. Bagley has shown, both experimentally and clinically, that the presence of blood in the subarachnoid spaces may, in the early stages, give rise to symptoms of meningeal irritation, and may, subsequently, by the formation of adhesions, interfere with the normal circulation of the cerebrospinal fluid, leading ultimately to a type of external hydrocephalus. Therefore, in those cases showing blood in the spinal fluid, as much fluid as possible was withdrawn, and lumbar punctures were done at daily intervals until the fluid became grossly clear. From a perusal of Table IV, it may be readily seen that lumbar punctures were performed more than twice as often in 1932 as in 1927.

Table IV
SPINAL PUNCTURE

	1927		1932	
	Cases	Per Cent	Cases	Per Cent
No. of cases in which spinal puncture was *not* done	30	55.5	4	6.0
No. of cases in which spinal punctures were done	24	44.5	56	94.0
Character of fluid				
Clear	3	12.5	5	9.0
Slightly bloody	4	16.6	17	30.0
Moderately bloody	10	41.7	19	34.0
Markedly bloody	7	29.2	15	27.0
Degree of pressure				
Normal	7	29.2	31	55.0
Slightly increased	4	16.6	7	13.0
Moderately increased	10	41.7	15	27.0
Markedly increased	3	12.5	3	5.0

In the latter year, only 5 cases had more than one spinal puncture done, while in 1932, 66 per cent of the cases had more than one spinal puncture, the number of punctures performed on each patient varying between two and ten.

The use of intravenous glucose solution serves a twofold purpose. Firstly, it is an excellent procedure in combating the preliminary shock, and secondly, it produces a definite and marked lowering of the intracranial pressure by withdrawing fluid, by osmosis, from the brain into the blood stream. The usual procedure in these cases was to give 50 c.c. of a 50 per cent solution, and repeat it as often as seemed necessary, but in general two injections daily for the first two to four days was sufficient. Other intravenous solutions, such as hypertonic saline and magnesium sulphate, were not used in this series due to their reported more toxic properties. In fact, Milles and Hurwitz have recently shown by animal experimentation that while glucose does produce a fall in intracranial pressure, there is a subsequent rise in pressure after several hours to a height greater than before its use. However, in our experience, the use of intravenous glucose has been quite satisfactory.

We have felt that one of the most important parts of the treatment of these cases is the fluid limitation. It is an established fact that if large amounts of fluid are given, the fluid after absorption will find its way to the brain, where it will be rapidly elaborated into spinal fluid, and thus add to the degree of intracranial pressure already existing. Therefore, in the 1932 series of cases, the fluid intake was limited to from 300 c.c. to 1000 c.c. in each twenty-four hours, dependent upon the age and size of the patient, the average intake for an adult being approximately 800 c.c. When this rule was strictly adhered to, it was found that after five or six days very little spinal fluid was elaborated. In those cases, however, in which there was a moderate or marked amount of blood in the spinal fluid (61 per cent), it was the custom to increase

slightly the fluid intake in order to wash out the blood cells. The importance of fluid limitation is so well illustrated in one instance in the 1932 series that it seems advisable to briefly report this case.

The case in point was that of a young man, aged twenty-one, who was admitted to the hospital in a state of moderate shock and unconsciousness. The spinal fluid was markedly bloody, and under a moderate increase in pressure. *X*-ray examination revealed an elongated fracture of the occipital bone on the right side. The reaction to shock measures was satisfactory, consciousness returned after several hours, and he was mentally clear at the end of thirty-six hours. On the third day after entry, his condition was satisfactory, with a pulse of 90, but he complained bitterly of thirst. In spite of repeated warnings, the patient drank the entire contents of a large ice bag. Within four hours he complained of intense headache, became extremely restless, and it was found that the pulse had dropped to a rate of 64. An immediate lumbar puncture was done which was productive of 25 c.c. of spinal fluid under a moderate increase in pressure. Within one and one-half hours, the patient was again resting comfortably with a pulse rate of 84. The subsequent convalescence was uneventful.

It is important to remember, however, that too drastic limitation of fluids may be harmful and often dangerous in certain cases. We have felt that the pulse rate is the most reliable guide in this matter; the fluids being limited in the presence of a normal or slowed pulse rate, and forced in cases showing a rising pulse, particularly when the rate is 120 or more.

Magnesium sulphate, given orally or by rectum, was used almost routinely in this series of cases as a further aid in the dehydration process. Whenever possible, this drug was given an hour or more after the use of the intravenous glucose, at a time when the blood stream showed an optimum fluid concentration, on the theory that the magnesium sulphate would be most effective at this time as a dehydrating agent.

So much for the routine treatment of these cases. The current literature indicates a divergence of opinion in regard to operative indications in skull fracture cases. We are inclined to agree with Fay and other authors that in general there are only two conditions in which surgery is indicated.

Firstly, in compound or depressed skull fractures. Obviously, marked depressions should be elevated, but this is best done after a delay of several hours or days when the condition of the patient is such that he can withstand surgery with a greater margin of safety. In the reviewed series of 1927 and 1932 there were 4 such cases in each series. In 1927 one case of compound fracture of the skull terminated fatally within three hours without operation. A second case of compound fracture, due to a gunshot wound, had an uneventful convalescence without operation. There were, in addition, 2 cases showing small (size of quarter) depressed skull fractures which recovered without resort to surgery. In 1932, 2 patients with extensive compound, comminuted skull fractures died in less than two hours after entry to the hospital without surgery being attempted. A third compound fracture, in this instance due to a gunshot wound, recovered with a simple debridement being done. One depressed skull fracture was observed in this series which terminated favorably following an elevation of the depressed area.

Secondly, surgery is indicated in cases of extradural hemorrhage due to middle meningeal bleeding, or in cases giving symptoms of subdural hemorrhage with localizing neurological signs. Middle meningeal hemorrhage is frequently difficult to diagnose particularly in the early stages and, according to Frazier, 50 per cent of the massive middle meningeal hemorrhages are overlooked. In the present survey of cases, no operation was performed for extradural hemorrhage in either series of cases. A review of the fatal cases occurring in 1927 reveals the fact that in

two instances large extradural hemorrhages were described at post-mortem examination, but in each case the outcome was so rapidly fatal that surgery could not have been contemplated. There were also in this same series 2 patients surviving nine and fifty-four hours respectively, who upon retrospect might have been benefited by an exploratory decompression operation, but unfortunately permission for autopsy was not granted and the presence or absence of extradural hemorrhage could not be determined. Among the cases terminating fatally in 1932, there was only one proved case of extradural hemorrhage. The interesting point in connection with this case was that in spite of a large extradural and smaller intradural hemorrhage, there was no evidence of blood in the spinal fluid at the time of lumbar puncture.

CONCLUSIONS

A total of 114 cases of craniocerebral injury was reviewed, only cases of proved skull fracture or brain laceration being included. The first series, numbering 54, comprised all such cases entering the Denver General Hospital during the first eight months of 1927, while the second series, numbering 60, included all those cases admitted during the corresponding months of 1932. The mortality in the former was 31.5 per cent, and in the latter 28.3 per cent, a difference of 3.2 per cent.

A survey of the 1932 cases reveals the fact that they were of a more serious nature than those of 1927. The accuracy of this statement is readily recognized when we consider: first, that 8 per cent more patients during 1932 were admitted to the hospital in an unconscious state; second, that the period of unconsciousness lasted three times as long in 1932; and third, that complicating or associated injuries of a grave nature were more frequent in this year. Furthermore, excluding from this series the 3 cases previously described in which death was obviously due to causes other than the skull fracture, the mortality for 1932 is 24.6 per cent, or a reduction of 6.9 per cent from the mortality rate of 1927.

The treatment in 1927 was simply supportive, while the treatment in 1932 was directed toward reducing the cerebral edema and increased intracranial pressure by means of frequent spinal punctures, intravenous concentrated glucose solution, limitation of fluids, and the use of magnesium sulphate. We are firmly convinced that this so-called dehydration therapy is the treatment of choice in skull fractures, and we believe that without this type of therapy, the mortality in the latter series of cases would have been appreciably higher. To support this belief, we find that the average survival period before death supervened was found to be twice as long in 1932 as in 1927, which might be interpreted as indicating that by the present mode of treatment the length of life was at least prolonged, and perhaps in the future more of these cases might be saved.

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NEUROGENIC IMBALANCE OF THE PELVIC ORGANS

TREATMENT WITH SYMPATHETIC GANGLIONECTOMY*

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THE advances made in the investigations of the autonomic nerves supplying the pelvic organs have brought forth a new form of therapy in certain disease conditions.

The following case is reported because of the vivid application of these principles and to stress the importance of surgery in carefully selected cases.

HISTORICAL REVIEW

Attention was directed toward pelvic neuralgia, megacolon and vesical disorders in 1898, when Jaboulay first performed the operation to interrupt the sacral sympathetic chain by a retrorectal approach. About one year later, Ruggi published his work on abdominal sympathectomy in functional disorders of the female genital organs. Then Leriche (1921) introduced periarterial sympathectomy as a means of relieving pain in cases of functional dysmenorrhea and in 1925 Cotte found that section of the presacral nerve gave complete relief in this type of case.

Hunter and Royle (1924) reported beneficial results in spastic paraplegia. Brown and Adson, during the same year, reported the first cases of lumbar sympathectomy and ganglionectomy in Raynaud's disease. In 1930, Learmonth and Braasch made a preliminary report of resection of the presacral nerve in the treatment of cord bladder. In 1932, Fontaine and Hermann reported 28 cases of pelvic plexalgia in which 22 cases were relieved by section of the presacral nerve.

ANATOMY

The inferior mesenteric plexus gives rise to the right and left mesenteric nerves which unite below the origin of the inferior mesenteric artery to form the presacral nerve (superior hypogastric plexus). In a number of cases, we have observed a branch from the lumbar chain. The branches from the left side pass obliquely downward while the branches from the right pass posterior to the artery. There are usually many fine anastomoses between these nerves so that a true plexus is formed. This plexus is situated anterior to the bifurcation of the aorta, and separated from it by a thin layer of fascia. The plexus then follows the curve of the sacrum into the pelvis. Anteriorly the plexus is covered by a thin layer of loose connective tissue and the parietal peritoneum. The root of the mesentery of the sigmoid colon is situated to the left.

There are many variations of the superior hypogastric plexus but none of vast importance. Latarjet and Bonnet (1913) gave it the name of "presacral nerve." In about 20 per cent of the cases, the nerves join to form a true nerve, and in 80 per cent, the nerves are more or less spread out in plexus formation (Delmas and Laux).

The few filaments from the superior hypogastric plexus course along the internal iliac arteries. The plexus itself continues down over the promontory of the sacrum and at the level of the first sacral vertebra divides into two distinct nerves, the inferior hypogastric nerves.

The inferior hypogastric nerves course downward into the lateral rectal space and terminate into a mass of nerve fibers and ganglion cells named the hypogastric ganglion which is situated on the superior pelvic space between the peritoneum and the fascia covering the levator ani muscles.

* From Mercy Hospital, Des Moines.

This plexus forms again on the anterior surface of the rectum just posterior to the uterus and vagina, and becomes intimately connected with the uterosacral ligaments.

CASE REPORT

An unmarried woman, twenty years of age, complained of inability to void or defecate except after taking 2 or 3 oz. of castor oil. The bladder and bowel would empty every four days and this was associated with extreme pain. This condition had persisted for two years and was also associated with a severe dysmenorrhea.

The past history was negative except that the menses had not appeared until she was eighteen and that for three years her hands and feet were cold, blue and clammy. During extremely cold weather, the toes and fingers would blanch and become very painful.

The general physical examination was negative except for the cold, clammy, cyanotic extremities and a mass in the abdomen which extended 4 cm. above the umbilicus. The patient was admitted to the neurosurgical service at Mercy Hospital on October 14, 1932, and after catheterization, the abdominal mass disappeared. At this time, 1000 c.c. of urine were withdrawn. Examination of renal function was normal, and upon cystoscopy, the bladder appeared normal except for its atony and marked distention. Roentgenograms of the colon revealed a moderately dilated colon with a large loop in the sigmoid but with no evidence of obstruction. Neurological examination, and spinal puncture at the fourth lumbar interspace were negative for evidence of subarachnoid block. Wassermann tests of the blood and spinal fluid were negative as were the other laboratory tests except for a small amount of albuminuria.

The patient was not considered to be suffering from hysteria, and with the presence of a Raynaud's disease, it seemed significant of an autonomic imbalance.

On October 21, 1932, under ether anesthesia, a midline incision was made, the hypogastric plexus was removed and a bilateral lumbar sympathetic ganglionectomy was performed. Immediately after operation, the lower extremities were warm, dry and pink. The surface temperatures of the feet were 28°C. before operation and after surgery they were 33°C.

Four hours after returning from the operating room, the patient voided 150 c.c. of urine with no difficulty. It was necessary to administer enemata for seven days before a spontaneous bowel movement occurred with the aid of mineral oil. However, the patient was able to void three and four times daily after the operation. On the fifth postoperative day, the menses appeared with no distress. The patient made an uneventful recovery and was dismissed from the hospital on the sixteenth postoperative day.

When last seen on January 15, 1932, the patient stated that she had been voiding three and four times daily, and there was a daily bowel movement without pain. The feet have remained warm, dry and pink, and each menses was practically painless.

SUMMARY

This case is illustrative of pelvic plexalgia and the inhibitory role played by the sympathetic nerves in the function of vesical and bowel evacuation.

Such a gratifying result is an added stimulus toward further investigation of the physiology of the autonomic system in the hopes that other surgical procedures may afford relief in carefully selected cases.

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[For Remainder of References see p. 475.]

NEUROGENIC APPENDICITIS

A STUDY OF 195 CASES OF APPENDICULAR NEUROMAS*

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THIS study is based on 344 consecutive vermiform appendices removed at operation showing no gross and microscopic evidences of acute or subacute inflammation. This study attempts to determine the incidence and what symptom complex, if any, these appendical neuromas (so-called neurogenic appendicitis) may show. In the experience of the busy surgeon, abdomens may have been opened with the preoperative diagnosis of acute or subacute appendicitis or acute exacerbation of a chronic appendicitis; yet much to his surprise, he may have found an appendix showing no gross evidence of an acute inflammation. This study attempts to find out if such an appendix may be the seat of neuromatosis giving rise to the acute symptomatology.

It is well known that there is no definitely established specific pathology for so-called "chronic appendicitis," since clinical chronic appendicitis has been found at operation and after microscopic study to be due to a variety of different conditions. Butka,[1] who studied 202 consecutive cases of appendectomy, believes that "chronic appendicitis" must be frequently functional and is more of a clinical than a pathological entity. Frequently, too, the cause of the discomfort and pain in the lower right quadrant is not due to lesions in the appendix itself. A long, mobile and dilated cecum may be the real cause of pain and tenderness at McBurney's point, due to the pulling and stretching of the mesenteric nerves (Wilms[2]). Lane's ileal kink and Jackson's pericolic membrane may give similar symptoms.

It is also well known that symptoms simulating acute inflammatory appendicitis may be caused by non-suppurative and non-bacterial conditions, such as infestation by the oxyuris vermicularis, endometriosis, and tumors of the appendix itself. Among the extra-appendiceal causes of acute symptoms are non-specific and tuberculous mesenteric lymphadenitis, strangulated epiploic appendage, and diaphragmatic pleurisy.

All the symptoms of acute or chronic appendicitis may arise from the presence of oxyuris vermicularis. Yet at operation, the appendix may be normal in appearance. Short[3] operated on 15 cases with a diagnosis of acute febrile appendicitis and on removing an apparently normal appendix showed that each contained oxyurides. Hence, he advises the operator who has qualms as to his diagnosis to examine carefully the interior of the appendix for these thread worms. In this series of 344 appendices, thread worms were found in 4 specimens.

Endometriosis of the vermiform appendix may sometimes be diagnosed as acute appendicitis or acute exacerbation of a chronic appendicitis, if this condition is not kept in mind. Diagnosis need not be difficult if one remembers that the acute pain in the right lower quadrant coincides with or is made worse during menstruation, because these ectopic endometrial tissues actively participate in the menstrual phenomena of hyperplasia, congestion, hemorrhage and exfoliation. Both of Hosoi and Meeker's cases[4] presented symptoms of appendiceal colic definitely related to the menstrual periods. Meigs' patient[5] was

* From the Department of Surgery, Tulane University School of Medicine, New Orleans, La. Abstract of thesis, presented in partial requirement for the degree of Master of Science in Surgery, Tulane University.

operated on for acute appendicitis; the appendix, bound down in dense adhesions, was found to have endometriosis microscopically. In Seelig's case[6] of endometriosis of the appendix, the woman suffered from acute abdominal pain, more or less indefinitely referred to the right iliac fossa where there was exquisite tenderness over McBurney's point but no rigidity; all laboratory findings, including temperature and pulse, were negative. Of the 344 appendices examined by us, there was one with implantation endometriosis of the serosa. At the time of operation, the finding of blue-domed endometrial implants or the presence of thick bloody fluid in the pelvic cavity will indicate the proper diagnosis.

Freeman[7] writes that pain of chronic non-specific mesenteric lymphadenitis is frequently colicky in character, occurring around the center of the abdomen with a strong tendency toward the right iliac fossa. Tenderness and rigidity are also usually present. As Alvarez[8] puts it, almost every symptom and sign will point sometimes to the presence of acute appendicitis and yet when the abdomen is opened, the surgeon is embarrassed to find no sign of disease; but if the surgeon would always look carefully in the mesentery of the lower part of the ileum, he not infrequently would find there a remarkable enlargement of the lymph nodes. Gage[9] has paid special attention to these mesenteric node enlargements and microscopically has found only a picture of hyperplasia; so far, cultures of these nodes on suitable media under aerobic and anaerobic conditions have been negative. Also, tuberculous mesenteric lymphadenitis (tabes mesenterica) in children and young adults may provoke a frequently unavoidable diagnosis of acute appendicitis and yet the appendices may be normal. Time and again, it has been emphasized that children with diaphragmatic pleurisy or early basal pneumonia have been operated on with the mistaken diagnosis of acute abdomen because of the presence of pain, tenderness, hyperalgesia and muscular rigidity in the right iliac region. In the very recent contribution of Palmer and Hardman,[10] attention is again focused on strangulated epiploic appendage; its diagnosis is very difficult and the condition has been most commonly confused with acute appendicitis.

Benign tumors of the appendix itself will also give symptoms of acute appendicitis. Miescher's patient[11] was seized with severe colicky abdominal pains which later localized in the lower abdomen. There was tenderness in the ileocecal region. Operation revealed a tumor (having the anatomical location of the appendix which itself was not found), which histologically proved to be a plexiform angiofibroma. Koontz's case[12] of fibromyoma of the appendix showed clinical symptoms of cramp-like upper abdominal pain which gradually became localized at McBurney's point. Physical examination showed considerable tenderness at McBurney's point. The leucocyte count was normal. It is interesting to note that one-half of the 14 cases of myomas collected by Koontz presented symptoms suggestive of appendicitis and were operated upon because of these symptoms. There were no cases of fibromas or myomas of the appendix in this series of 344 consecutive appendices. So rare are these tumors that they may be eliminated in the differential diagnosis of the neuromatous appendix. Carcinoid tumors of the appendix may give rise to symptoms suggestive of ordinary acute or chronic appendicitis. These tumors usually occur at the tip of the appendix and may signify their presence at the time of the operation by a bulbous enlargement. There were 2 cases of carcinoid tumors in this series of 344 appendices. Compared with the incidence of neuromas of the appendix, carcinoids are rare indeed. Masson,[13] who has done an immense amount of work on the origin of these carcinoids, concludes that carcinoids result from the autonomous proliferation of the isolated neurocrine or argentaffin cells which he found are also always present in appendiceal neuromas.

Furthermore, he found that these neuromas occur in the axial region of completely obliterated appendices in 86 per cent of the

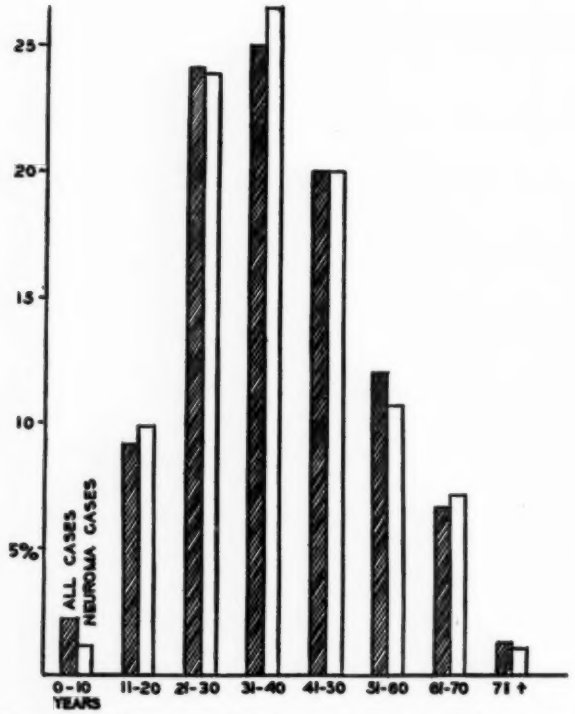

Chart 1. Histogram showing percentage age frequency of neuromas of appendix as compared with all consecutive appendices removed at operation, showing no evidence of acute or chronic inflammation.

specimens and that these tumors arise from the periglandular nerve plexus.

Examination of 195 Cases of Neuromas of the Vermiform Appendix

Three hundred forty-four consecutive vermiform appendices, removed at operation and showing no evidence of an acute inflammation, were studied microscopically, serially and otherwise. One hundred sixty-five or 48 per cent had a distally or totally obliterated lumen. There were 195 cases or 56.7 per cent of neuromatosis of the appendix. Of these 195 cases, 135 or 69.2 per cent occurred in obliterated appendices and 60 cases or 30.8 per cent in patent ones. This gave an incidence of 82 per cent of neuromatous appendix for all obliterated appendices.

Description of the Neuromas: There was nothing characteristic grossly to indicate the presence of appendiceal neuromatosis. When the neuromatous growth was grossly noticeable, its cut surface appeared opaquely grayish-white, usually without well-demarcated borders. The larger growths measured not more than 1.5 to 2 mm. in diameter.

The number of neuromatous growths varied from one to many. When multiple, they were linked together by many nerve fiber strands. They appeared as loosely anastomosing strands of non-medullated nerve fibers or as islands of compact interlacing bundles of spindle-shaped cells, located always central to the muscularis mucosae. If the neuromatous tumor mass was large enough, the muscularis mucosae was closely apposed to it along the periphery in the manner of a capsule. Rarely the tumor mass broke through this muscularis mucosae. These neuromatous growths contained a variable admixture of either argentaffin cells or lymphocytes.

Age: Royster[14] observed that between ten and thirty years is by far the most common period for the occurrence of ordinary appendicitis. Because obliteration of the lumen begins normally about the age of thirty, appendicitis of the inflammatory type is not found to be so common after this age. As shown in Chart 1, the age incidence of the neuroma cases follows so closely the age grouping of all cases that age appears to have no special etiologic importance. The extremes in ages were eight and seventy-nine years.

Sex: From the literature of acute appendicitis, Hosoi and Alvarez[15] found men were a little more affected; on the other hand, they found that "chronic appendicitis" was diagnosed more frequently in women because the appendix was so often removed during the course of operations on the pelvic organs. Klotz[16] studied 5647 appendices and found that the obliterated appendix occurred almost three times as frequently in women as in men. This is what we found in this series of 344

consecutive appendices including the 195 appendiceal neuromas, not only because the appendix was removed during pelvic operations but also because female patients predominated even where the appendix alone was removed.

Symptomatology: *Pain:* This varied from severe cramp-like pains to mere discomfort. In 16 or 8.2 per cent of the 195 neuroma cases, the cramps were so intensely severe that these patients doubled up or rolled about in extreme agony, simulating a perforated peptic ulcer. In 37 others, or 19 per cent, the pains were severe but not so extreme as in the foregoing group. These described their pains as being severe, sharp, considerable, knife-like, stabbing or griping in character. These acute pains were as a rule momentary or intermittent. They may be generalized all over the abdomen, or start in the epigastrium, umbilical region, or right lower quadrant, to be followed by a more or less dull ache localized in the right lower quadrant, which may then be constant in duration. The pain starting in the right lower quadrant may radiate to the epigastrium, umbilical region, or across the midline slightly into the left lower quadrant and then radiate back again to the right lower quadrant. This makes a total of over 27 per cent in which the pains were severe enough to cause much suffering. In 15 or 7.7 per cent, there was definite pain in the epigastrium, right lower quadrant or sometimes in the right flank. In those cases where the pain was in the right flank or high up in the right lower quadrant and radiated to the back, the appendix at operation was found to be retrocecal. Some of these patients with retrocecal neuromatous appendices complained of pain and tenderness in the right costovertebral angle.

In 20 patients or 10.3 per cent, there was a complaint of dull ache in the right lower quadrant, which was constant, intermittent, or even periodic. In some of these cases, there were sometimes dull aches, at other times sharp pains. In others, the dull ache became a persistent "gnawing" or "nagging," or increasingly worse so that definite pain was experienced.

Only a discomfort in the right lower quadrant or epigastric distress was present in 6 or 3.1 per cent. In the remaining 101 or 51.8 per cent, no symptoms or signs referable to the appendix could be obtained in the past history because they were usually masked by other more important acute or more insistent chronic complaints. These 101 patients came to the hospital for acute symptoms of cholecystitis, peritonitis, intestinal obstruction by omental bands, and ectopic pregnancy; and for chronic symptoms of multiple leiomyomata uteri, chronic pelvic inflammatory disease, endometriosis, uterine and ovarian malignancy, large ovarian cysts, relaxed pelvic floor, procidentia, cholecystitis and cholelithiasis, duodenal ulcer, hernias, malignancies of the stomach and rectum, and spinal cord lesions.

Nausea and Vomiting: Nausea alone was present in 25 or 12.8 per cent; vomiting alone in 5 cases or 2.6 per cent; both nausea and vomiting in 21 cases or 10.8 per cent; or a total of 26.2 per cent where either one or both were present. The nausea varied from slight and mild to considerable and severe. The number of vomiting spells varied from one to many. Besides these symptoms, three others complained of having epigastric distress, "indigestion," or of being "sick in the stomach." In one case, the very first symptom was a sudden attack of much vomiting.

Tenderness: This was elicited in 76 cases or 39 per cent. This varied from slight to considerable. Most of this tenderness was elicited just above, just below, or exactly at McBurney's point but sometimes the whole right lower quadrant was tender with the maximum point of tenderness at McBurney's. In one case, there was one point of tenderness in the midline suprapubically. In 8 cases, this tenderness was present only on deep pressure at or near McBurney's point. The tenderness was exquisite or marked in 18 cases or 9.2 per

cent. There was definite rebound tenderness in 3 cases. Psoas test was positive in 2 cases.

Right Rectus Rigidity: There were 29 cases or 14.9 per cent in which right rectus rigidity was present. It was slight in 9 cases, moderate in 9, and marked in 11. In one of these cases, the entire abdomen was rigid throughout with the patient rolling about in agony, simulating some abdominal catastrophe.

Temperature, Pulse and Respiration: In all those cases where the symptoms were definitely referable to the appendix with no other pathology present elsewhere, the temperature was normal or slightly subnormal except in 11 cases where it was around 99°F., including 3 cases at 100°F. In these last 11 cases, the pain in the right lower quadrant tended to be severe.

The pulse was normal in 50 per cent of the cases, 81 to 90 per minute in 27 per cent, and 91 to 100 per minute in 23 per cent.

The respirations were normal in over 77 per cent of the cases, 21 to 24 per minute in 22 per cent. In one case where the respiration was 28 per minute (see Case VII), the temperature and pulse were also elevated.

White Blood Count: Unfortunately, in the great majority of cases, no white blood and differential counts were obtained. In those cases where these were made, the white counts and differentials tended to be normal (see Case Reports). In 2 cases where the total white count exceeded 10,000 (see Case XXXIV), the differential count was within normal limits; in Case XXVII, there was no record of a differential count being made.

Constipation and Diarrhea: There was constipation of varying degrees in 36 cases or 18.5 per cent. Constipation alternated with diarrhea in 2 cases. In another case, there was no constipation but considerable diarrhea during the attacks of acute right lower quadrant pain. Thus, it is seen that neurogenic appendicitis may either tend to inhibit intestinal activity or stimulate it, the former to a much greater extent than the latter. Some of the patients complained of gaseous distention of the abdomen with the expulsion of much flatus; others belched more or less.

REPORT OF REPRESENTATIVE CASES

The following 50 case reports represent the various combinations of acute and chronic symptoms and signs these cases of appendiceal neuromas (neurogenic appendicitis) may show. In every case, one or more neuromas were demonstrated microscopically. In no case was there any gross and microscopic evidence of acute or chronic inflammation in the appendix.

Although there has been no follow-up in any of these cases, at the time of discharge from ten to fourteen days after operation the patients had complete relief from all previous complaints. According to the record department, there was no record of any of these patients returning to the hospital during the two years this study has been in progress.

First Group: This group of 30 cases includes those having severe cramp-like pains, simulating acute appendicitis or an acute exacerbation of a chronic appendicitis. Observe in the very first case that although the symptomatology and physical examination point toward an acute appendicitis, yet the normal total white and differential counts and the normal temperature would be opposed to such a diagnosis.

Case I. L. P. V., male, twenty-eight years old. The day previous to his admission to the hospital, he was taken with severe pain around the umbilicus which radiated to the right lower quadrant. He described it as feeling as though something was pulling on his navel from the inside. He was extremely nauseated and had to lie down. There was considerable pain and distress all night. On the morning of admission, the pain was localized in the right lower quadrant.

When he was ten years old, he had a similar attack, which subsided in a few days under treatment by a physician. Over six months ago, he had another colicky attack.

Physical examination showed marked tenderness in the right lower quadrant, both on pressure and rebound. Some tenderness was also elicited in the right flank. There was definite muscle spasm in the right lower quadrant. No palpable mass was felt in the abdomen. Rectal examination gave tenderness on the right side. Urinalysis was negative. The white blood count was 5900 with a differential of 68 per cent polymorphonuclear leucocytes, and 32 per cent lymphocytes. Temperature, pulse and respiration were normal. He was operated on with the diagnosis of acute appendicitis, but only an obliterated appendix was found. There was no other pathology found in the abdominal cavity.

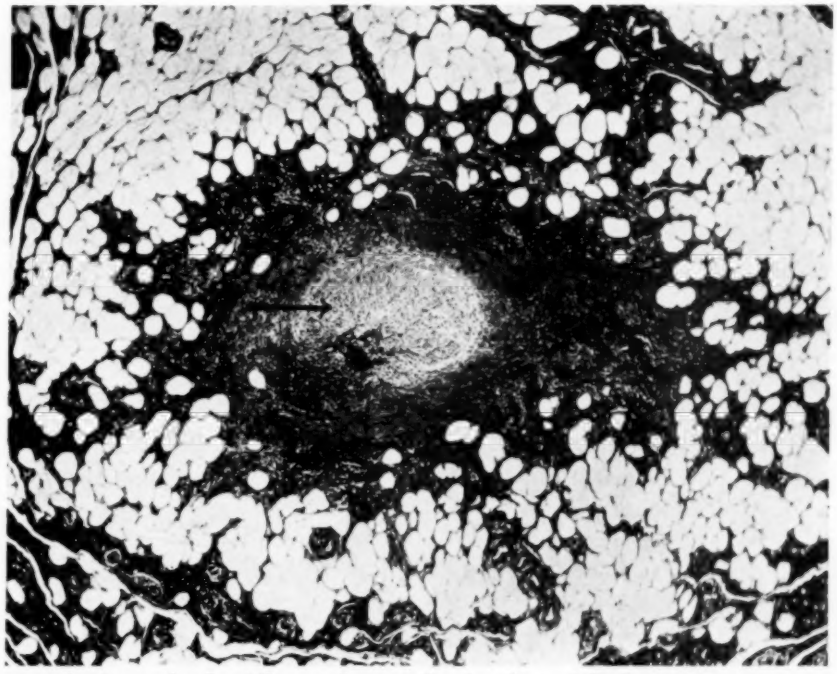

Fig. 1. Photomicrograph of completely obliterated appendix showing axially located neuromatous growth (arrow). Note interlacing bundles of nerve fibers. Mallory's phosphotungstic-acid hematoxylin. × 41.

Case II. H. B., female, fifteen years old. About seven days ago, while recovering from a scarlet fever attack, she was taken with severe epigastric distress. Pain was rather diffuse. She vomited five times. Next day, pain was considerably better. Though quite uncomfortable, she has not had severe attacks again. Upon physical examination, there were some muscular resistance and tenderness in the right lower quadrant with the greatest tenderness at McBurney's point. Urinalysis was negative. White blood count was 8200 with a differential of 70 per cent polymorphonuclear leucocytes, 14 per cent lymphocytes, and 16 per cent large mononuclear leucocytes. Temperature was normal. At operation, a grossly normal appendix was removed; there were no abnormal findings in the abdomen.

Case III. A. B., female, fourteen years old. Three weeks ago, while at school, she suddenly developed nausea and vomiting, followed by severe generalized cramping abdominal pain, which later gave place to more constant right lower quadrant pain. She was in bed for one week, when pain and tenderness gradually subsided. She thought she had fever at that time. She had relief from lying on her right side with thighs flexed. She returned to school two weeks ago, and has had no return of acute symptoms but considerable right lower quadrant dull pain and tenderness, made worse by exercise. She was not constipated. The right lower quadrant showed tenderness and muscle spasm. On rectal examination, the right side was tender. Urinalysis was negative. White blood count was 8200. Temperature, 99°F., pulse, 84, and respiration, 20. Preoperative diagnosis was subacute appendicitis; operation showed the appendix to be patent, not inflamed, and not adherent. The pelvic organs were normal.

Case iv. B. B., female, twenty-one years old. She has suffered from much nausea for the past five months, but without vomiting. She had two acute attacks during this period; the first attack occurred suddenly five months ago while taking a bath, lasting about five minutes. The second attack came on one week ago and was much worse, lasting from ten to fifteen minutes. Each attack consisted of griping pain in the pit of the stomach, shifting to the right lower quadrant. Her appetite was poor, constipation frequent, cathartics being necessary every three or four days. Examination showed only a tenderness in the right lower quadrant. Urinalysis was negative. Temperature 98.4°F., pulse 95, and respiration 18. The appendix was found to be free and patent throughout, the pelvic organs being grossly normal.

Case v. S. W., male, twenty-three years old. He had rather an uncomfortable feeling in his abdomen all afternoon. Then at 6 A.M., while eating his breakfast, he was seized with marked epigastric pain which was severe enough to double him up. There was nausea but no vomiting. It eased a little and he walked to his work. Here, he was seized with another attack, worse than the previous one and this time the pain radiated to the right lower quadrant. By the time of admission to the hospital, the pain had disappeared. On examination, the right lower quadrant was rigid, with distinct tenderness at McBurney's point. Urine showed a change in color for sugar. Temperature 98.5°F., pulse 94, and respiration 23. At operation, a non-adherent, non-inflamed, patent appendix was found; there was no other pathology elsewhere in the abdomen.

Case vi. E. C., female, eighteen years old. For the past three weeks, the patient has had considerable pain in the right side. This pain first started in the right side, lasting for a day or so. It then went to the epigastric region and later returned to the right side. There has been no vomiting until two days ago, when she became nauseated and vomited. Her bowels have been regular. During the last day or so, the pain has been worse. Examination showed tenderness and spasm over McBurney's point. Temperature was normal. Urinalysis was negative. At operation, the appendix was patent and non-adherent. (See Fig. 4.)

Case vii. E. C., female, twenty-three years old. For the past year, she has had attacks of pain in the right lower quadrant, which occurred every three or four weeks. At times, the pain was so severe that it doubled the patient up, but this did not last more than five or ten minutes. She has had a great deal of nausea and vomiting, which occurred with the pain and also independently of it. For the last three days, she has vomited everything that she ate. The last severe pain occurred three days ago. There was tenderness over McBurney's point but no spasm. Urinalysis showed a definite trace of sugar. Temperature 99.4°F. pulse 92, respiration 28. At operation, the appendix was patent with no adhesions; uterus and adnexae and the gall bladder were normal.

Case viii. T. T. E., male, twenty-six years old. He has had pain in the right lower quadrant off and on for the last five days. This pain was dull and lasted a couple of days each time. Five days ago, he began having a rather severe pain in the right lower quadrant but did not have any nausea or vomiting. The pain was steady and grew gradually worse up to the time of admission. There was no diarrhea or constipation. The right lower quadrant was tender and rigid. Urinalysis showed a few white blood cells. Temperature 98.5, pulse 83, respiration 20. The preoperative diagnosis was subacute appendicitis. At operation, the appendix was non-adherent, patent, and not inflamed; the other abdominal organs were normal.

Case ix. C. D., female, thirty-one years old. A year ago, she was seized with a dull cramping pain in the right lower abdomen, which lasted three days. She was nauseated but did not vomit. Since then, she has had two similar attacks. Four days ago, a fourth and most severe attack of all occurred with much nausea but no vomiting. She had to stay in bed. Physical examination showed definite McBurney point tenderness and moderate spasm of the right rectus. There was tenderness in the right vault on bimanual examination. Temperature, pulse, and respiration were normal. Urinalysis was negative. The appendix removed at operation was of the obliterated variety. There was no pelvic pathology. (See Figs. 2A and B.)

Case x. M. S., female, twenty-eight years old. She has had irregular attacks of abdominal pain, accompanied by nausea for the past two and one-half years. Three weeks ago she had a severe attack of abdominal pain, coming on about 1 A.M., and lasting until 6 A.M., with a residual right lower quadrant pain and tender-

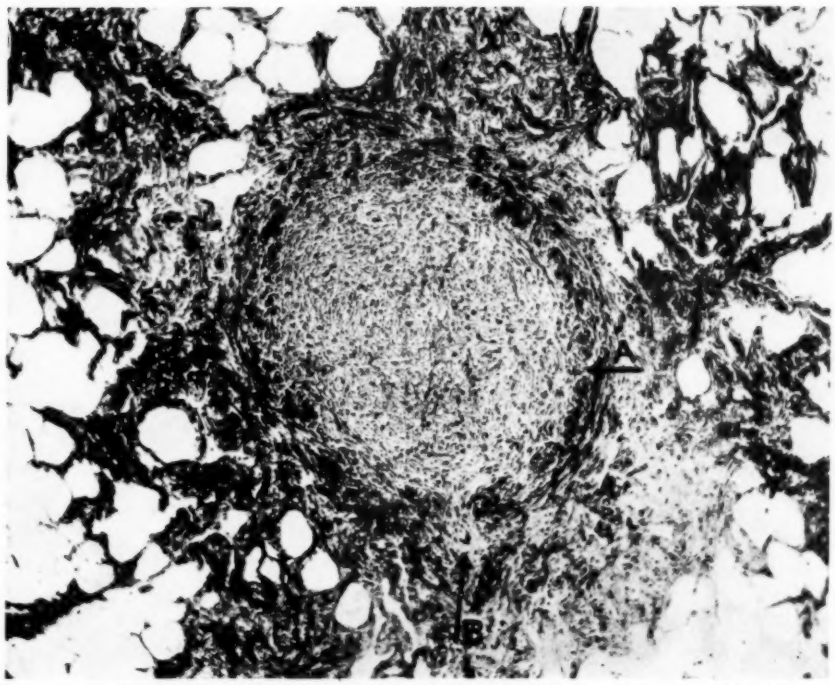

FIG. 2*a*. Photomicrograph of another well-developed axially located neuroma surrounded by muscularis mucosae in capsular fashion (A). At one point (B), neuroma has broken through muscularis mucosae and is extending into submucosa. Mallory's phosphotungstic-acid hematoxylin. × 134.

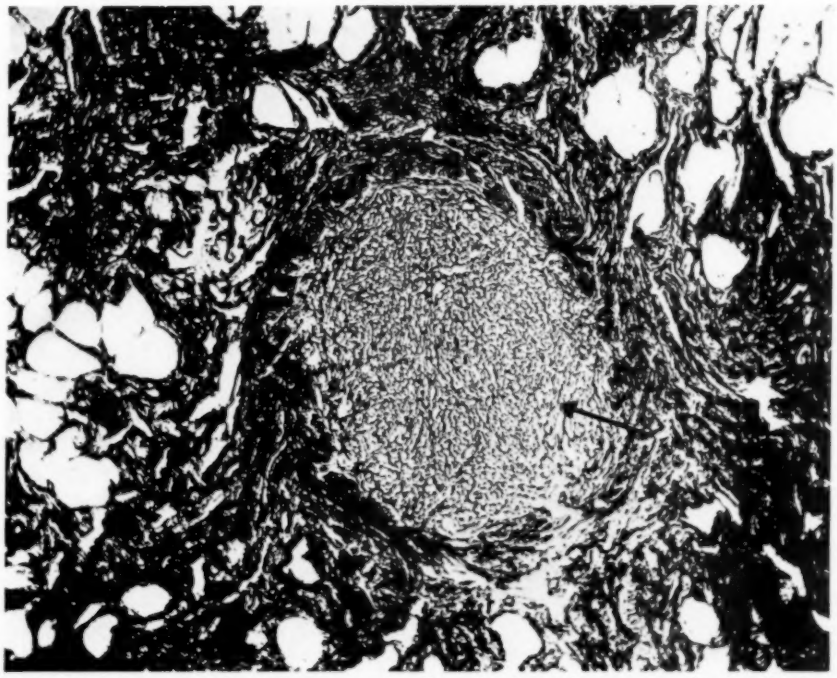

FIG. 2*b*. Photomicrograph of same section of neuroma to show intricate lacy network of reticulum (arrow) forming a supportive tissue for nerve fibers. Laidlaw's silver stain. × 134.

ness lasting three days, accompanied by nausea but no vomiting and no fever. There has been marked constipation for the past two and one-half years. Her right lower quadrant showed some slight tenderness and rigidity. Urinalysis was negative. Temperature 98.2°F., pulse 80, respiration 25. Operation showed a patent, non-adherent appendix; her pelvic organs were normal.

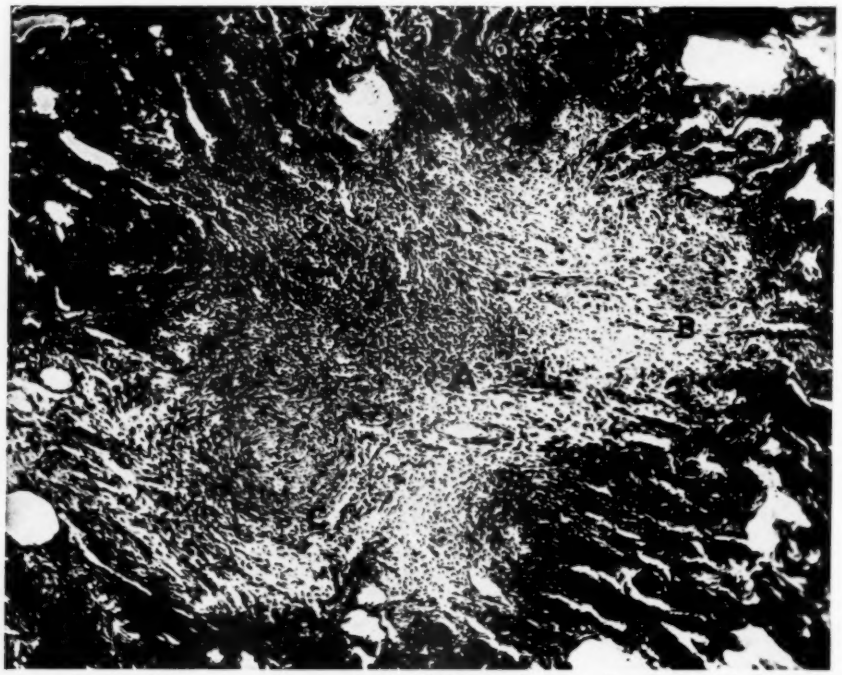

FIG. 3. Photomicrograph of axial portion of completely obliterated appendix showing infiltration of lymphocytes (A) into neuromas to indicate retrogression of these neuromas. Even in this section, many nerve fibers are still present, running out from neuromas (B, C). Serial sections show that these nerve fibers run from one neuromatous nodule to another. Masson's trichrome. × 134.

CASE XI. B. C., female, thirty-seven years old. Following an attack of grippe, she developed severe abdominal pains. At first, the pain was low in the right lower quadrant just above the inguinal ligament; later, it was slightly higher. Was in bed for seven weeks and has had some pain ever since. Examination showed tenderness in both lower quadrants most marked on the right, with a good deal of right lower quadrant rigidity. Urinalysis was negative. Temperature 98°F., pulse 95, respiration 20. The appendix was found to be completely obliterated, without adhesions. (See Fig. 1.)

CASE XII. M. K., female, twenty-four years old. Two weeks ago, at the end of a day when she had not felt well and had not worked, she felt a sudden sharp pain in the right lower quadrant. She doubled up with pain. This gradually subsided so that she was able to walk back home. Since then, she has been having almost continuous "grumbling" pain in that region. She was somewhat nauseated but did not vomit. There was no diarrhea. For the past two years, she has suffered with "chronic appendicitis," characterized by severe pains, not so sudden and severe as in the present attack, in the right lower quadrant with nausea. These pains were more apt to come on during menstrual periods. She has had indigestion for many years with pains in the epigastrium and nausea at times. She always was constipated. The right lower quadrant was tender. Urinalysis was negative. Temperature 97.5°F., pulse 80, respiration 19. The appendix at operation was found free and patent. The pelvic organs were normal.

CASE XIII. E. J., female, twenty-one years old. She had pain in the right side for the past two weeks, and was nauseated for most of this time, vomiting frequently almost every day, Pain started in the pit of the stomach but two days later became localized in the right side. She was in bed two or three days at a time, but would get up in between times and do housework. When the pain first started, it was severe

and cramp-like, but later became an aching sensation. Examination gave tenderness in the right lower quadrant. Urinalysis was negative. Temperature was normal. The preoperative diagnosis was subacute appendicitis. At operation, the right tube and ovary were normal; the appendix showed no adhesions and the lumen was patent.

Case xiv. H. F., female, twenty-seven years old. Last night at about 10:30 p.m., she became conscious of pain in the abdomen about the umbilicus. She went to bed and slept till 4 a.m., when she awoke with very severe pain about the umbilicus which shifted to the right lower quadrant. Pain continued and was still very severe. She had felt nauseated since pain started but did not vomit. On physical examination, the abdomen showed a slight but definite spasm of the right rectus, marked tenderness over the right lower quadrant, extending to the midline and slightly to the left. Rebound sign was positive. Psoas sign elicited slight tenderness. Urinalysis was negative. Temperature 98.8°f., pulse 96, respiration 22. The preoperative diagnosis was acute appendicitis. At operation, the only pathology found in the abdomen was a non-adherent, obliterated appendix.

Case xv. M. R., female, thirty years old.

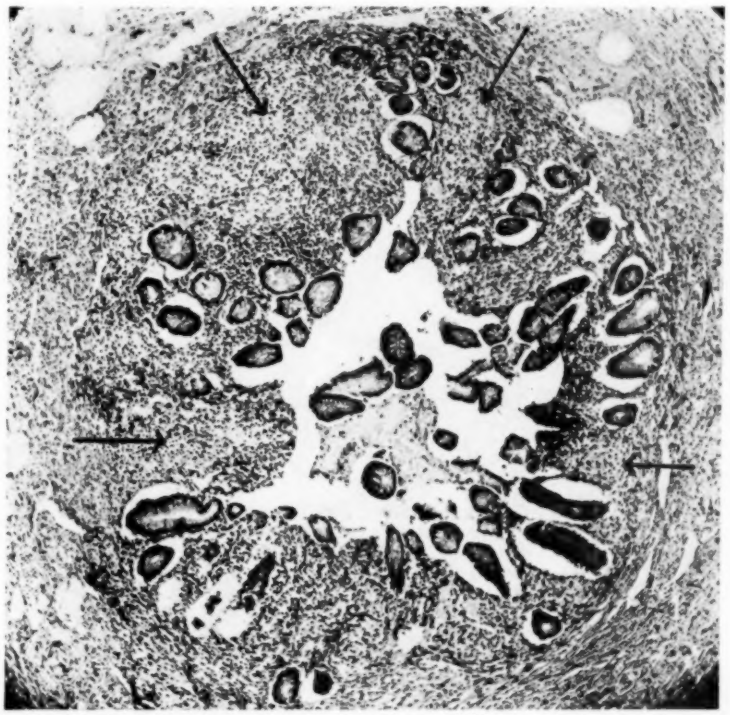

Fig. 4. Photomicrograph, showing that lumen of appendix is still patent but undergoing obliteration, due to atrophy of glandular elements and marked decrease in lymphoid cells. Mucosa in this section contains numerous neuromas (light areas, some of which are indicated by arrows). Hematoxylin and eosin. × 115.

Four times during the past year, at intervals of about two or three months, she has had attacks of quite sharp pain in the right lower quadrant. Generally, they started about 2 a.m., and lasted from three to four hours. There were no associated nausea, vomiting or constipation; no urinary symptoms, and no relation to menses. During the intervals between these attacks, the patient was practically free from pain but was conscious of a very slight ache in this region. The last attack was four days ago. Physical examination showed considerable tenderness in the right lower quadrant, just above McBurney's point and extending downward toward the pelvis. Urinalysis was negative. Temperature 98.6°f., pulse 88, respiration 24. Preoperative diagnosis was subacute appendicitis but at operation only an obliterated appendix without adhesions was found; no other pathology was present.

Case xvi. P. D., female, twenty-three years old. One evening some days ago, had occasional attacks of pain in the right lower quadrant.

The pain would last for a few minutes and then disappear. There was no nausea or vomiting. Since that time, at irregular intervals, the pain has been present, and has always localized in the right lower quadrant. On the morning of admission to the hospital, it became much more severe and the patient was obliged to go to bed; was slightly nauseated but did not vomit. Patient was most comfortable lying with thighs flexed. Physical examination showed marked tenderness and spasm in the right lower quadrant. The left side of the abdomen was also somewhat tense. Urinalysis was negative. Temperature, 98.8°F., pulse, 81, respiration, 20. Preoperative diagnosis was subacute appendicitis. At operation, the uterus and adnexae, gall bladder and ileum were normal. The appendix was free but obliterated.

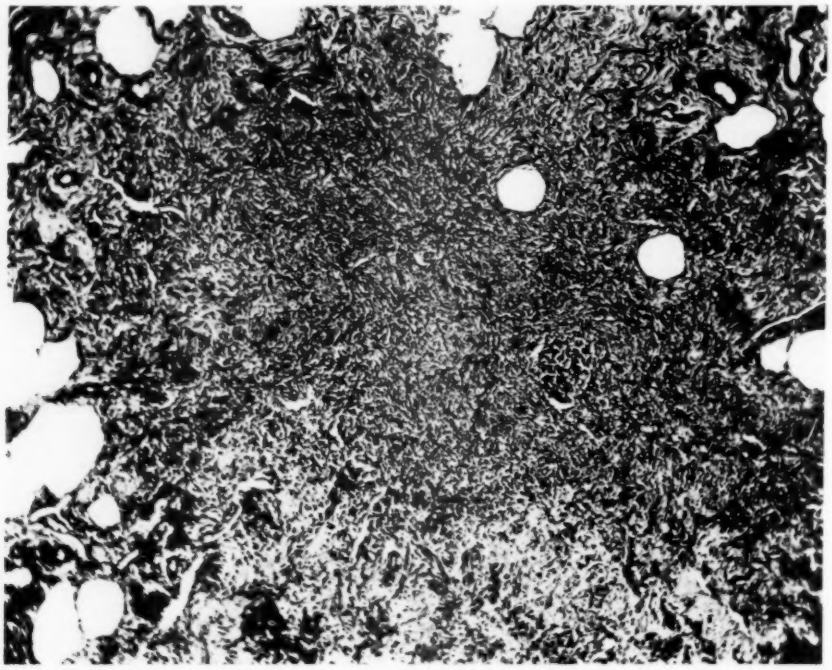

Fig. 5. Photomicrograph of axial portion of a completely obliterated appendix showing general absence of muscularis mucosae and centrally located, ill-defined mass of neuromatous tissue. Lymphocytes are beginning to invade neuroma. Masson's trichrome. × 133.

Case xvii. E. B., female, twenty-eight years old. This patient gave a history of acute pain beginning two weeks previously in the right lower abdomen. This acute attack subsided and was replaced with a dull ache for the past two weeks. On physical examination the abdomen was flat with moderate tenderness on deep pressure in the right lower quadrant and slight spasm of the right rectus. Urinalysis was negative. Temperature, pulse, and respiration were normal. Although this appendix was patent throughout, its lumen was extremely narrow and the mucosa markedly atrophic.

Case xviii. D. W., female, thirty-two years old. The patient has been annoyed with attacks of pain in the right lower quadrant for many years. These attacks were either associated with or were independent of her menstrual periods. Of late, they have become more severe and were definitely accentuated when they occurred during menses. In the recent attacks, there was some nausea but no vomiting. She was constipated. There were no urinary symptoms. Examination showed only some tenderness as in the right lower quadrant. Urinalysis was negative. Temperature 98.8°F., pulse 65, respiration 20. An obliterated appendix with no adhesions was found at operation.

Case xix. B. H., female, seventeen years old. Three months ago, while playing basketball, the patient was kicked in the abdomen, which became diffusely tender. Three weeks ago, she fainted while dancing. This was followed by a constant, marked pain and tenderness in the right lower quadrant. There was no nausea or vomiting. A few days ago, she was seized with sharp pain in the right lower quadrant which was constant. There was no

vomiting but some nausea. When she moved, the pain became worse. The right abdomen was markedly tender and spastic, especially in the right lower quadrant. Movements of the lower extremities produced pain in the right lower quadrant. There was no rebound tenderness. Urinalysis was negative. Temperature 99.2°F., pulse 88, respiration 19. The preoperative diagnosis was subacute appendicitis. At operation, the pelvic organs were normal; the appendix was non-adherent and non-obliterative.

Case xx. M. B., female, seventeen years old. During the past four years, the patient has had several attacks of pain in the right lower quadrant, each attack lasting a week or so. It was a dull ache with intervals of rather sharp and acute pain, at times going somewhat to the back. There was no nausea or vomiting till the last attack. These attacks usually confined her to bed for a time. The last attack was about two weeks ago and this time she had some nausea but no vomiting. Also, the pain radiated down into the right leg and the patient said that the right foot became somewhat swollen. There was no dysuria or other urinary difficulties, no change in bowel habits with the attacks and no constipation. She felt well between attacks. Physical examination showed tenderness on deep pressure in the right lower quadrant. Urinalysis was negative. Temperature, pulse and respiration were normal. Operation showed the right tube and ovary to be normal; the right ureter was small and no stones were felt anywhere. The appendix was normal grossly and the lumen contained no oxyurides.

Instead of constipation, these cases sometimes have diarrhea, as illustrated in Cases xxi, xxii, xxx and xlix. In Case xxi, the diarrhea alternated with constipation. The loose movement may occur only once, or many times. This may or may not be coincident with the abdominal pain. Even in the severe cases, the diarrhea stopped at the most about the third day. Case xlix is interesting in that the patient suffered from a morning diarrhea with two or three bowel movements before he finished dressing.

Case xxi. M. K., female, twenty years old. One month ago, she had a severe attack of pain in the right lower quadrant, with considerable nausea and vomiting. Constipation alternated with diarrhea. The attack was severe for one to two days and gradually lessened. Since then, she has had occasional slight aches in this region on overexertion or stretching. On examination, both lower quadrants were slightly tender, less so on the left. Urine showed a slight trace of albumin. Temperature 97.8°F., pulse 78, respiration 20. Operation revealed a normal uterus and adnexae; the appendix was free with a patent lumen.

Case xxii. K. H., female, twenty-one years old. At 6:30 A.M. today, she was awakened by pain in the lower abdomen, more marked in the right lower quadrant. This was severe and was accompanied by nausea, but no vomiting. She had a loose bowel movement following this. She has had previous attacks of pain in the right lower quadrant but without the acute manifestations of the present illness. Temperature, pulse and respiration were normal. At operation, the pelvic organs were normal to palpation; the appendix was free, with the tip slightly clubbed, and the lumen obliterated.

Case xxiii. H. L., female, twenty-five years old. She entered the hospital with the chief complaint of severe cramp-like abdominal pain, more pronounced in the right lower quadrant. The pain came on suddenly and became progressively worse until her admission. She vomited once. For the past three days, she has had a general digestive upset and became constipated. She had a similar attack two months ago. Examination showed great tenderness in the lower abdomen, more marked in the right iliac region. Temperature 98°F., pulse 100, respiration 23. Urinalysis was negative. At operation, the appendix was grossly normal in size and shape, but was completely obliterated, and bound down by adhesions. The gall bladder, stomach, fallopian tubes and ovaries were normal. (See Fig. 5.)

Case xxiv. C. S., male, fifty years old. Last night about 7 P.M., he began having severe pain in his abdomen at about the level of the umbilicus. The pain was very intense and sharp in character. He said that the pain caused him to roll about. He vomited but did not remember the type of vomitus. Pain continued, becoming more severe, and remaining localized near the umbilicus just to the right of it. At midnight, he came to the hospital. He

had had two previous attacks similar to this, although with less intense pain, eight and ten years ago, respectively, was treated conservatively both times with ice packs and was sick for three to four days on each occasion.

Physical examination showed a pale, emanciated male in acute pain. The abdomen was rigid throughout and very tender, with the greatest tenderness on the right side near the umbilicus. There were no palpable masses. Urinalysis was negative. Temperature 98°F., pulse 93, respiration 20. The preoperative diagnosis was acute appendicitis with peritonitis. The peritoneal cavity contained greenish fluid. No pathology was found in the stomach, intestines, gall bladder and liver. The appendix was of the non-adherent, obliterative variety. Recovery was good.

Cases XXV, XXVI, XXVII and XXVIII in the first group and Cases XLVIII and XLIX in the second group showed a retrocecal appendix at operation. Both Cases XXV and XLVIII complained of pain in the right costovertebral region in addition to the pain in the right lower abdomen, anteriorly.

Case XXV. E. B., female, thirty-eight years old. For the past two years, the patient has had attacks of pain starting in the epigastrium and localizing in the right lower quadrant. The pain was severe but without nausea or vomiting. These attacks occurred every few months and lasted until the doctor gave some medicine for relief. She never had diarrhea but was always constipated. She usually felt ill for a few days with the attacks, then recovered completely. The last attack was three or four weeks ago, similar to those previous ones, but this time the pain has continued in the right lower quadrant. She said the pain this time occurred also in the right costovertebral angle and right leg, and pointed to a sore spot rather high in the right lower quadrant. She has had indigestion for a long time, giving an indefinite history of gas and epigastric distress after meals; and sometimes pain after meals all across the epigastrium. She was never jaundiced and had no tarry stools.

On physical examination, there was tenderness in the right costovertebral angle and over the whole right side anteriorly but most marked just at McBurney's point. Urinalysis was negative. Temperature 99°F., pulse 80, respiration 20. At operation, an obliterated, retrocecal appendix was found; there was no other pathology.

Case XXVI. L. K. N., female, twenty-seven years old. About seven years ago, the patient had an attack of pain and tenderness in the right lower quadrant. There was no fever, chill, or vomiting but she was in bed for one week. She was told she had "appendicular colic." For the past year, she has had occasional nausea with pain in the epigastrium, relieved by food or soda. For the past three months, she has had pain in the right lower quadrant, sometimes severe and sharp, but mostly dull. It sometimes interfered with her work and sleep and she had to go to bed for a day or so which relieved her. Often she had no pain for a week or more but no fever at any time. The bowels were regular. There were no urinary or menstrual irregularities. On physical examination, there was some tenderness in the right lower quadrant on deep pressure. Urinalysis was negative. Temperature, pulse and respiration were normal. At operation, only a retrocecal, obliterated appendix was found.

Case XXVII. H. B., female, sixty-two years old. The patient has had some intermittent dull pain in the right lower quadrant for the past six months, but no previous acute attacks. Last evening, she developed severe pain in the same region, with nausea and vomiting. The pain was severe, crampy and relieved by flexing the right thigh. It was duller this morning, but considerable local soreness was still present. Although there was some tendency toward constipation, the bowels moved normally this morning. The right lower quadrant was slightly tender but not spastic. Urinalysis was negative. White blood count was 12,500. Temperature 98.6°F., pulse 88, respiration 20. At operation, an obliterated appendix was found retrocecally. The pelvic organs were normal.

Case XXVIII. F. T., female, twenty-three years old. Three years ago, she first noticed pain in the right lower quadrant, relieved by pressure on the abdomen. Since then, at varying intervals for the last two years, momentary knife-like pain was felt in the lower quadrant region, whenever she rolled over on her right side, especially in the morning on waking. Two weeks ago, while returning on a train, she felt a sudden sharp pain in the right lower quadrant, which went to the midline. She had a sudden desire to urinate. The pain continued

and subsided somewhat when the train stopped. Soda gave some relief. The next morning, had another sharp right-sided pain. There was considerable epigastric discomfort. There was some tenderness in the right lower quadrant, especially at McBurney's point, and in the midline in the lower abdomen. Urine showed a color change for sugar, and a faint trace of albumin. Temperature 98.6°F., pulse 89, respiration 20. Operation revealed a retrocecal patent appendix. The pelvic organs were normal.

The following 2 cases and Case L show that the diagnosis of neurogenic appendicitis may be difficult and impossible. In Case XXIX, the history did not give any localizing symptoms and signs in the right lower quadrant. In Cases XXX and L, although the symptomatology pointed to some pathological condition in the right lower quadrant, the physical examination was entirely negative. These patients are very apt to be labeled neurasthenic or hysteric, especially highly-strung women.

Case XXIX. R. S., female, twenty-two years old. About five months ago, she had an attack of generalized abdominal pain accompanied by nausea but without fever. Two weeks ago, there was a second attack, similar to the first but more severe; this time, the pain, chiefly in the mid-abdomen and above the umbilicus, was so severe as to cause her to double up. It was accompanied by nausea and vomiting. She was in bed for one day, and thought she had fever with this attack. The patient felt well at the time of admission to the hospital. There have never been any localizing symptoms in the right lower quadrant. There was some tendency to constipation, controlled by mineral oil. The appetite was good. However, examination showed very slight rebound tenderness in the right lower quadrant, and tenderness on deep pressure in the midline slightly below the umbilicus. Urinalysis showed a very faint trace of albumin. Temperature 98°F., pulse 80, respiration 20. At operation, the pelvic organs and other abdominal organs were normal; the appendix was non-adherent and patent, not inflamed.

Case XXX. P. G., female, twenty-five years old. For five years, she had been suffering from occasional attacks of pain in her right side. Two months ago, she had an acute attack of right lower quadrant pain, lasting about an hour and gradually subsiding. There was no vomiting but considerable nausea and considerable diarrhea. Since then, she has had almost constant dull aching pain in this region with occasional diarrhea and considerable nausea but no vomiting. The abdominal examination was entirely negative. Urinalysis was negative. Temperature 97.9°F., pulse 80, respiration 20. At operation, the appendix was free, its lumen patent; the pelvic organs showed no pathology.

Second Group: This group of 20 cases includes those with complaints of dull aching, soreness, distress or discomfort, simulating an ordinary chronic appendicitis. This aching or soreness was usually dull and either constant, intermittent or occasional; however, it became worse in some cases so that temporary acute pain was experienced. It usually had no relation to menstruation but sometimes the onset of menses made the ache worse.

Case XXXI. J. L., male, thirty-eight years old. About five weeks ago, he began to have some discomfort in the abdomen, likened to gas pains. He noticed a considerable amount of pain in the right side when he lifted objects. There was some nausea but no vomiting, no food distress, some relief after bowel movement. He had some discomfort two years ago in the same region with some constipation. On deep palpation, some tenderness could be elicited in the right lower quadrant with slight tenseness and point tenderness at McBurney's point. Urinalysis showed a very slight trace of albumin and few white blood cells. White blood count was 7150. Temperature 98.7°F., pulse 98, respiration 20. Operation revealed a patent appendix without adhesions; no pathology was found in the abdomen.

Case XXXII. M. M., female, twenty-seven years old. She has had pain in the right lower quadrant for the last four weeks. There was no nausea or vomiting. Examination showed considerable tenderness over McBurney's point but no spasm. Urinalysis was negative. Temperature 98°F., pulse 78, respiration 20. At operation, the right tube and ovary were normal; the appendix was free and patent.

Case XXXIII. G. H., female, twenty-one years old. For the past two years, she has had

a dull aching pain in both lower quadrants, more so on the right side; this last year, she has been conscious of this ache most of the time. There were no real acute attacks, no nausea or vomiting and no urinary symptoms. The chronic constipation seemed to be worse when the aches got bad. The onset of menses made the ache a little worse. There was slight tenderness in the right lower quadrant and even less in the other side. Urinalysis was negative. Temperature 99°F., pulse 88, respiration 23. At operation, the pelvic organs were normal; the appendix showed no adhesions but was obliterated.

Case xxxiv. J. C., male, twenty years old. One month ago, he felt a pain in his right lower quadrant. A cathartic somewhat lessened the pain, which however lasted for three days, to be followed by a comfortable period of several weeks. Five days ago, this pain and discomfort in the right lower quadrant returned; it was constant. He became constipated; there was no vomiting. Examination showed tenderness at McBurney's point. Urinalysis was negative. Temperature was 99°F. White blood count was 13,900 with a differential of 74 per cent polymorphonuclear leucocytes, and 26 per cent lymphocytes. At operation, only an obliterated, non-adherent appendix was found.

Case xxxv. L. W., female, twenty-seven years old. Five days ago, the patient began to have pain in the right lower quadrant, which was dull and constant, never sharp and severe. She did not sleep well that evening. This dull pain continued for the next two days. There was no nausea, vomiting, diarrhea, dysuria, chills or fever. The pain disappeared yesterday and has not come back since. In the past, there was occasional indigestion with pains in the lower abdomen, not at all severe. On physical examination, only a slight tenderness in the right lower quadrant was present. Urinalysis was negative. Temperature, pulse, and respiration were normal. The appendix was found to be completely obliterated but non-adherent. (See Fig. 3.)

Case xxxvi. P. M. S., female, twenty-six years old. For the past two or three months, she has had a dull aching pain in the right lower quadrant, which was intermittent, never acute and severe. There was no nausea or vomiting. However, the pain sometimes caught her quite sharply. It was not worse during menstruation. She was moderately constipated. Examination showed only a slight tenderness in the right lower quadrant. Urinalysis was negative. Temperature, pulse, and respiration were normal. The appendix on removal was of the obliterative type without adhesions. The pelvic organs were normal.

Case xxxvii. J. K., male, twenty-four years old. Eight months ago, he noticed fullness in the pit of his stomach and soreness in the right lower quadrant, accompanied by a dull, aching pain in both regions. Eating made conditions worse. There was slight nausea but no vomiting. He had lost 18 pounds in the last six months. A tendency to constipation for the past month was present. Physical examination showed tenderness on deep pressure in the pit of stomach and right lower quadrant. Urinalysis was negative. Temperature 98°F., pulse 75, respiration 18. At operation, the gall bladder and stomach were normal; the lumen of the appendix was patent throughout; there were no adhesions.

The next case and Case xxvi in the first group had a symptomatology strongly indicative of a peptic ulcer; yet at operation, the stomach, duodenum and even the gall bladder were found to be normal on exploration, both by inspection and palpation. These reflex symptoms emanating from some pathology in the appendix contribute to the protean nature of the symptomatology of appendicular disease.

Case xxxviii. J. B., male, thirty-four years old. For the past eight or nine years, the patient has complained of obstinate constipation. He suffered from persistent "gnawing" pain in the right lower quadrant but there was never any nausea or vomiting. He also has had gastric distress one hour after meals, accompanied by persistent eructations of gas and burning, relieved by alkalies. Physical examination showed tenderness in the right lower quadrant, more marked over McBurney's point with marked muscular spasm of the right rectus. Temperature 98.8°F., pulse 88, respiration 22. At operation, no other pathology but a non-adherent, obliterated appendix was found. The stomach and gall bladder were normal.

Case xxxix. M. M. H., female, twenty years old. For the past six years, the patient has had occasional attacks of pain in the right

lower quadrant, associated with nausea and vomiting. The pain has been rather dull but it interfered with her activities; she went to bed for two or three days with each attack. For the last two weeks, she has had frequent attacks of pain and has not been able to eat. On examination, there was only a very slight tenderness in the right lower quadrant. Urinalysis was negative. Temperature 98°F., pulse 72, respiration 18. At operation, the uterus and adnexae were normal; the appendix was obliterated and non-adherent.

Case XL. L. R., female, twenty-nine years old. She has had a "grumbling feeling" in the right lower quadrant off and on for eleven years. This was almost constant for the past two months, being much worse in the past month. This was associated with two attacks of nausea and vomiting, the first beginning two months ago and lasting one week, the second beginning three weeks ago and lasting two weeks. The right lower quadrant below McBurney's point was slightly tender. There was no rebound tenderness or spasm. Temperature 97.9°F., pulse 85, respiration 20. Urine showed a slight color change for sugar. At operation, the appendix was free and patent.

Case XLI. F. R., female, twenty-eight years old. The patient became constipated three days ago, for which she took a cathartic. This was followed by dull aches and pains, first in the epigastrium, later in the right side. There was no vomiting until the night of admission to the hospital. The right lower quadrant was tender and rigid. Urinalysis was negative. Temperature 98.5°F., pulse 82, respiration 18. At operation, the uterus was slightly enlarged and hard with normal adnexae and the appendix free and obliterated.

Case XLII. L. M. F., male, forty-two years old. About one and a half years ago, the patient began having discomfort in the right lower quadrant, consisting of some pain and gas but mostly an annoying aching and tenderness to palpation. He has been troubled considerably with gaseous distention of the large bowel. There has never been any nausea or vomiting nor eructations of large amounts of gas. These aching attacks have lately become more frequent until for the past two weeks, the patient has been almost completely disabled by the condition. He has chronic constipation. Examination elicited tenderness on moderate palpation over McBurney's point. Preoperative urine showed a slight change of color for sugar. Temperature 99°F., pulse 96, respiration 20. The appendix at operation was found to be almost completely obliterated and non-adherent.

Case XLIII. A. K., female, seventeen years old. About four years ago, she had her first attack of abdominal pain and several slight attacks since then. A few weeks ago, they returned. The pain was localized in the right lower quadrant, and associated with nausea but no vomiting or diarrhea. During the past few weeks, she has had several such attacks during which she had tenderness in the lower quadrants. These attacks of pain were worse if they occurred during a period and frequently followed menses. There was slight tenderness in the right lower quadrant on deep palpation. Urinalysis was negative. Temperature 97.5°F., pulse 75, respiration 20. Operation revealed a patent, non-adherent appendix. The pelvic organs were grossly normal.

Case XLIV. H. B., female, twenty-one years old. For the past four or five years, she suffered from periodic pain lasting a short time in the right lower quadrant, going around into the flank. There was no nausea. One week ago, she had a similar pain, lasting several days, with nausea but without vomiting. The abdomen was tender in the right lower quadrant at this time. Urinalysis was negative. Temperature 98°F., pulse 76, respiration 20. At operation, the pelvic organs were normal; the appendix was non-adherent and patent.

Case XLV. B. H., female, thirty-five years old. For eight years she had had a dull aching pain in the right lower quadrant, almost constantly present. No attacks of very severe pain, no nausea and no vomiting were ever present. Pain was not related to meals or menstrual periods. She was constipated and took a cathartic every night; otherwise, the pain in the right lower quadrant would become more severe. She has had a good deal of gas and some belching. The right lower quadrant over McBurney's point was tender and rigid. Urinalysis was negative. Temperature 98.8°F., pulse 91, respiration 22. At operation, the gall bladder and right tube and ovary were normal; the appendix was free, patent, and not inflamed.

Case XLVI. S. M. Y., female, thirty years old. In 1918, the patient had an attack of

"appendicitis" for which she was in bed for two weeks with pains in the right lower quadrant, nausea and vomiting but no fever. She had another but less severe attack in 1922 which kept her in bed for two days. About two weeks ago, she began to have a dull and constant pain in the right lower quadrant, which disappeared when the patient reclined. This has never been sharp and severe enough to interfere with work or sleep. She was slightly nauseated but did not vomit. Has been constipated lately. There was no fever or chills and no urinary symptoms. On examination, there was only a slight tenderness in the right lower quadrant. Temperature was normal. At operation, an obliterated non-adherent appendix was found.

The following Case XLVII is interesting in that the attack was initiated by a sudden onset of severe vomiting, followed later by a dull pain in the right lower quadrant. This variation in the sequence of events is not infrequently seen in other diseases of the vermiform appendix.

Case XLVII. M. P., female, thirty-five years old. Last week, she was suddenly seized with a vomiting attack, followed shortly afterwards by a dull "nagging" pain in the right lower quadrant. She had six such vomiting attacks on that day and the pain has been persistent. Since then, there has been no vomiting but a "sickly" feeling in her epigastrium. She has had no fever at any time. On admission to the Hospital, she was feeling very well. She has had similar attacks during the past three years, having no relation to menses. Examination showed some slight tenderness in the right lower quadrant, but no spasm. Urinalysis was negative. White blood count was 7200. Temperature 98°F., pulse 84, respiration 25. Operation revealed a non-adherent, patent appendix; the rest of the abdominal and pelvic organs were grossly normal.

Case XLVIII. H. M., male, thirty-two years old. At intervals for the last two or three years, the patient suffered from attacks of "indigestion" with epigastric distress with no nausea or vomiting. Eight months ago, when he strained himself, he felt a little sting in the right flank. Then about three weeks later, his "feeling of indigestion started again and kept getting lower, settling in the pit of his stomach." There was no nausea or vomiting. This lasted for about one month, during which time he was wholly incapacitated. Later, he experienced dull pain in the right costo-vertebral region, which remained fairly constant, but still there was no nausea or vomiting. His last attack, lasting four days, was over a week ago; the pain then was lower down on the right side and radiated to the navel. He felt bloated but there was no nausea or vomiting. He has always been constipated off and on. Tenderness on deep pressure was elicited in the right lower quadrant. Urine showed a slight change in color for sugar. The Wassermann reaction was negative. Temperature 98.5°F., pulse 80, respiration 24. At operation, the gall bladder, stomach and other viscera were found to be normal; the appendix was retrocecal and its lumen patent.

Case XLIX. H. G., male, forty-one years old. Two months ago, he began having some epigastric distress. This discomfort usually came on after breakfast and was burning in character. It did not last very long and did not recur after other meals. Food did not relieve this distress; he did not try soda. He was never nauseated and did not vomit. At times, there was some distention in the epigastrium. Occasionally, there was a morning diarrhea with two or three stools before he finished dressing. There were no chills, no jaundice, and no radiation of the discomfort to the back. Associated with his epigastric distress, he occasionally had pain in the right lower quadrant. For the last two to three weeks, he has had practically no epigastric discomfort while the lower abdominal pain has been getting more intense; this was never severe enough to double him up; it was usually dull but at times, sharp and stabbing. He was not constipated but took a cathartic once a week as he felt that this gave him some relief from his abdominal pain. Physical examination showed some tenderness at McBurney's point but no muscle spasm. Urinalysis was negative. Temperature 98.5°F., pulse 89, respiration 20. At operation, a retrocecal obliterated appendix was found; the other abdominal viscera were normal.

Case L. A. E., male, thirty-nine years old. There was a gradual onset of symptoms but with no nausea or vomiting. The pain was irregular in appearance, and was most noticeable at meal time. About a week ago, the patient was awakened from a sound sleep by

pain, dull in character, radiating from the right lower quadrant to the opposite side and back again. For the past few months, he also had associated pains in the right knee and thigh. These latter were made worse by walking so that he felt most comfortable while lying in bed. His appetite was good. He was always constipated. The abdomen showed no tenderness or spasm. Urinalysis was negative. Temperature 98.4°F., pulse 85, respiration 23. Operation revealed a non-adherent appendix high up with the cecum on the right side.

COMMENT

This study has shown that symptoms of severe or mild pain may arise from the presence of axial neuromas in the obliterated or non-obliterated appendix. Physical examination may even reveal a rigid abdomen, especially in the right lower quadrant, plus marked tenderness. In some cases, rebound tenderness was present and psoas test was positive. Their agony may be so extreme that these patients literally double up or roll about, trying to obtain relief; or their complaint may be simply a dull ache or discomfort in the right lower quadrant with only a slight tenderness at McBurney's point. Nausea with or without vomiting ranged from mild to severe and varied in number from one to many. In spite of the apparent acuteness of the symptoms and signs, the temperature is normal and the total white and differential counts appear to remain at normal levels.

At this point it may not be amiss to see if fibrosis may give rise to neuromatous changes elsewhere in the gastrointestinal tract. Stoerk's[17] patient, who had a gastrojejunostomy two years previously for gastric ulcer, complained of severe pains in the stomach region for the last six months. At the second operation, the chronic gastric ulcer was resected. On microscopic examination, the ulcer showed a neuromatous proliferation in the scar tissue portion, the structure of which was very much like that of amputation neuromas. Askanazy[18] reported similar observations of neuromas in the dense fibrous base of three different chronic gastric ulcers, in one of which ganglion cells were also present. He called these scar neuromas, which to a certain extent may be the cause of gastralgias. Okkels[19] who studied 34 gastric ulcers by means of serial sections found that the nerves were increased in number and size, running in all directions, and that enormous masses of nerve tissue were present in the ulcerated gastric wall. Hamperl's[20] case is most interesting. This man, sixty-two years old, suffered for about twenty years from gallstone colics with jaundice once or twice a year. Operation showed a shrunken gall bladder with stones in it and also in the hepatic and common ducts. Microscopic examination of its fibrotic wall showed an extraordinary neuroma-like formation, resembling the neuromas found in fibrosed appendices.

SUMMARY

1. This study is based on 344 consecutive vermiform appendices removed at operations, showing no gross or microscopic evidences of acute or chronic inflammation. Forty-eight per cent of all appendices had a partially or completely obliterated lumen.

2. There were 195 cases or 56.7 per cent of axial neuromas, proved microscopically. In a few instances, the neuroma produced a clubbing of the tip of the appendix. After proper fixation and staining by the Masson method, 69.2 per cent of these neuromas were found to occur in obliterated appendices and 30.8 per cent in non-obliterated ones. The incidence of neuromas for all obliterated appendices would then be 82 per cent.

3. Age appears to have no etiologic importance. The youngest patient was eight years old and the oldest seventy-nine years. The largest number of neuroma cases occurred between the ages of twenty-one and fifty, which was also the age incidence for all appendectomies.

4. No definite conclusions can be drawn as regards the importance of sex from this series of cases.

5. All gradations from severe cramp-like pains to mere discomfort were present in

the different cases. In 8.2 per cent, the pains were cramp-like and agonizingly severe. In 19 per cent, the pains were still acute not so extreme. In 7.7 per cent, there was definite pain but not acute. In 10.3 per cent, there was a complaint of dull ache in the right lower quadrant. Three and one tenth per cent complained only of a discomfort in the right lower abdomen or epigastric distress.

6. These pains may start as a generalized abdominal condition or in the epigastrium or in the umbilical region, later shifting to the right lower quadrant. Or, it may start in the right lower quadrant from the very beginning and radiate to the epigastrium, umbilical region or even to the back.

7. The acute pains were as a rule momentary or intermittent, to be replaced later by a dull ache. The milder symptoms of distress or discomfort or ache were either constant, intermittent or periodic.

8. Nausea alone was present in 12.8 per cent, vomiting alone in 2.6 per cent, both in 10.8 per cent; or a total of 26.2 per cent where either one or both were present. The nausea may be mild or considerable; the vomiting varied in number from one to many.

9. Tenderness, usually in the right lower quadrant, was elicited in 39 per cent and varied from slight to considerable. There was marked tenderness in 9.2 per cent. There was definite rebound tenderness in 3 cases and positive psoas test in 2 cases.

10. Fourteen and nine-tenth per cent showed right rectus rigidity. It was slight in 9 cases, moderate in 9 and marked in 11.

11. The temperature was normal in the great majority of cases (93 per cent).

12. The pulse was normal in 50 per cent of the cases, 61 to 90 per minute in 27 per cent, and 91 to 100 per minute in 23 per cent.

13. The respiration was normal in over 77 per cent of the neuroma cases, 21 to 24 per minute in 22 per cent.

14. The white blood and differential counts tended to be within normal limits. This and the normal temperature are important points in the differential diagnosis of non-suppurative (neurogenic) from suppurative appendicitis.

15. Constipation of varying degrees was present in 18.5 per cent. In a few cases, there was diarrhea which may alternate with constipation.

16. Before an appendix is pronounced microscopically negative for neuroma or neuromatosis, the Masson trichrome stain should be used.

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[For Remainder of References see p. 475.]

TECHNIQUE OF PHRENICECTOMY

USE OF A SHORT INCISION

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MUCH has already been written on the technique of the operation for removal of the phrenic nerve. No originality is claimed in this article. Its purpose is a plea for shorter incisions in this operation. It is entirely possible to avulse the phrenic nerve through an incision ½ inch long. Never should it be necessary to extend the incision to more than ⅞ inch. No difficulty is encountered with this length incision, and especially in women, a scar of such length is less noticeable than one three or four times as long (Figs. 7, 8, 9). The following is the technique employed in performing phrenicectomy through an incision of minimum length.

FIG. 1. Thin bladed steel retractors which encroach as little as possible on space within incision. They have been designed especially for operation of phrenicectomy and are an aid in performing operation through an incision of minimum length. Shanks are bent at different angles to provide for both lateral and medial retraction.

After the administration of a capsule of sodium amytal two hours preceding operation, and a hypodermic of morphine sulphate 1/6 grain and atropine sulphate 1/150 grain before being sent to the operating room, the patient is placed on the table reclining at an angle of about 30°. The face is turned away from the operator who stands on the affected side. After preparation of the skin and draping, the patient is asked to begin to raise the head with the face remaining turned away.

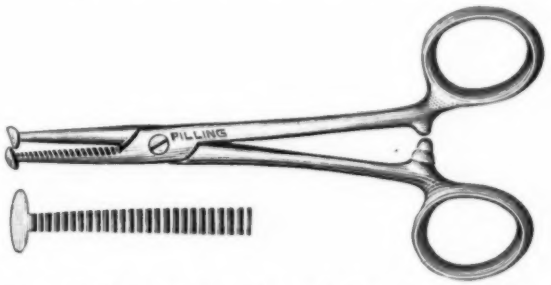


FIG. 2. Avulser used in this technique has a baffle at distal end which prevents nerve from slipping off instrument. There is no tip extending beyond baffle which may grasp fibers of scalene muscle in attempting to clamp nerve. Before nerve is clamped avulser should be passed through finger ring of an ordinary hemostat which then acts as a fulcrum for pull, as nerve is twisted about avulser.

The head need not actually be raised off the pillow, but the muscles are contracted as though this were to be done. This procedure puts into use the clavicular insertion of the sternocleidomastoid muscle (Fig. 3). The border can be easily palpated. The incision is to be made along the lateral border of this portion of the muscle. A solution of 1 per cent novocaine is then infiltrated in the skin beginning at a point one finger's breadth above the clavicle. The infiltration may continue for about an inch upwards following the border of the clavicular insertion of the sternocleidomastoid muscle. No attempt is made at infiltration of the deep tissues as anesthesia here is quite unnecessary.

An incision ½ inch (1.25 cm.) long is then made. The external jugular vein is thus avoided. It lies lateral and posterior, though parallel, to this incision. An advantage is gained here in averting the danger of air embolism, and time is saved in avoiding

ligation. The tissues are separated by blunt dissection using two plain thumb forceps. The platysma muscle is separated in the direction of its fibers. The cervical fascia is opened. One dissects bluntly into the tissues towards the anterior scalenus muscle which can be felt by placing the tip of the little finger in the incision. After penetrating the fascia, the retractors shown in Figure 1, which have been especially designed for this operation, are used. They are held by an assistant who stands on the opposite side of the table, holding the medial retractor with one hand and reaching across the patient to hold the lateral retractor with the other. The dissection continues down on a pad of fat. It is essential that the operator go directly through this pad of supraclavicular fat in order to come directly down on the anterior scalenus muscle in the normal location of the phrenic nerve. The retractors are

FIG. 3. An incision ½ inch long is made along lateral border of clavicular insertion of sternocleidomastoid muscle. External jugular vein is thus avoided.

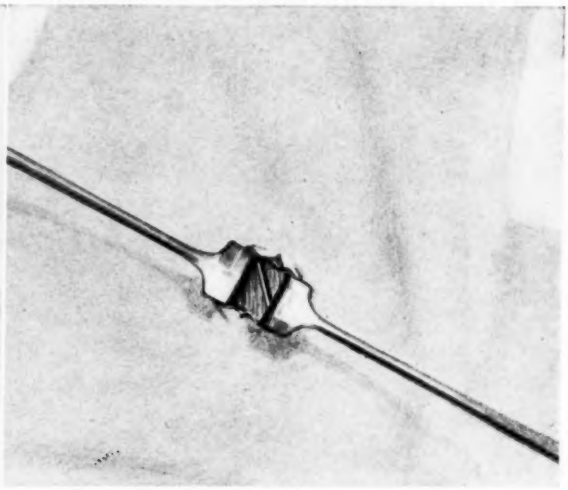


FIG. 4. Retractors do not encroach upon space within incision which is nearly perpendicular to direction of nerve.

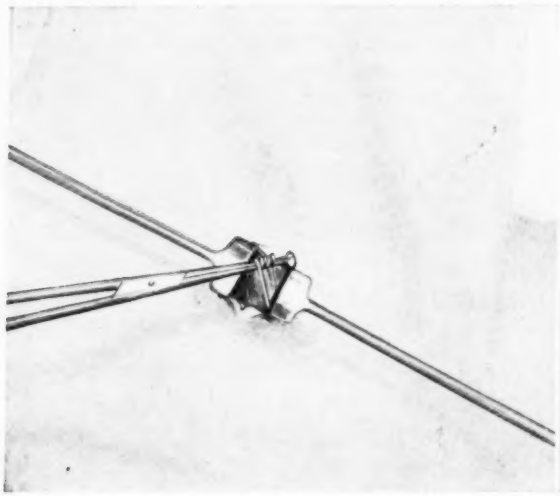


FIG. 5. Nerve is slowly twisted on avulser by rotation of latter. The slower twisting motion is done the more likely is operator able to obtain entire phrenic nerve. Rapidity in removing nerve is very apt to lead to breaking of fibers and failure in obtaining sufficient length of nerve to include collateral branches.

FIG. 6. Incision may be closed with one skin clip. Occasionally three clips may be placed close together, and if this is done, two of these should be removed day following operation, middle clip being allowed to remain in place for three days.

moved from time to time as the tissues are opened. Normally the phrenic nerve is found easily if one follows this technique,

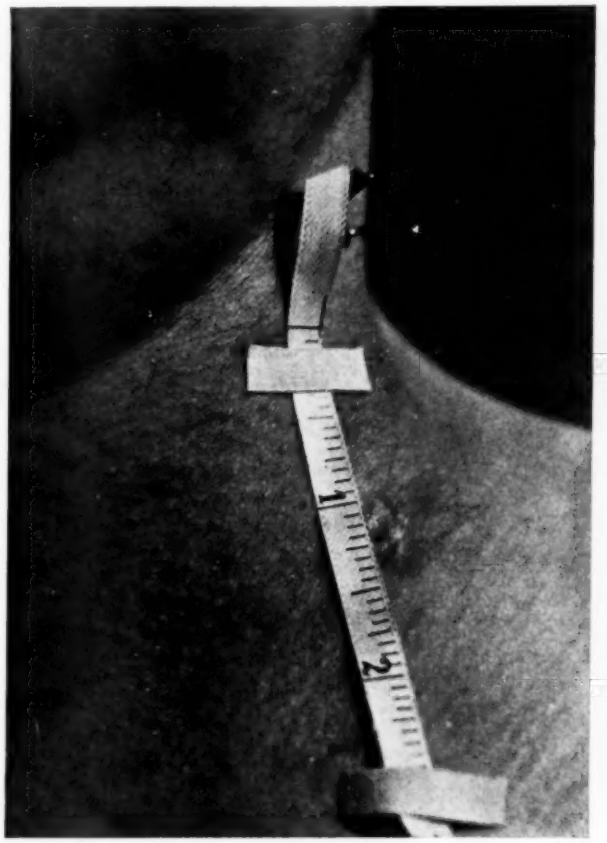


Fig. 7. Scar in this illustration, which has been taken with a magnifying lens, measures less than ½ inch. Length of incision day following operation was 1.25 cm. or exactly ½ inch.

and is located in its course running over the anterior scalenus muscle from the lateral to the medial side. The incision is nearly perpendicular to the direction of the nerve (Fig. 4).

The nerve is identified by grasping it gently with the thumb forceps. The patient complains of pain in the shoulder of the same side, and also in the chest on the same side. After identification, the nerve is freed from the surrounding areolar tissue and infiltrated high up with 1 per cent novocaine solution. The nerve is then clamped with the avulser shown in Figure 2, which is a modification of the one designed by Proctor. There is an advantage in not having a projection beyond the baffle of the avulser as the nerve is easily clamped without grasping fibers of the muscle. Before clamping the nerve, the shank of the

Fig. 8. Patient shown in illustration was well nourished and there was a good amount of subcutaneous fat in neck. It was still possible to avulse phrenic nerve, 11¾ inches being obtained, through an incision exactly ½ inch in length.

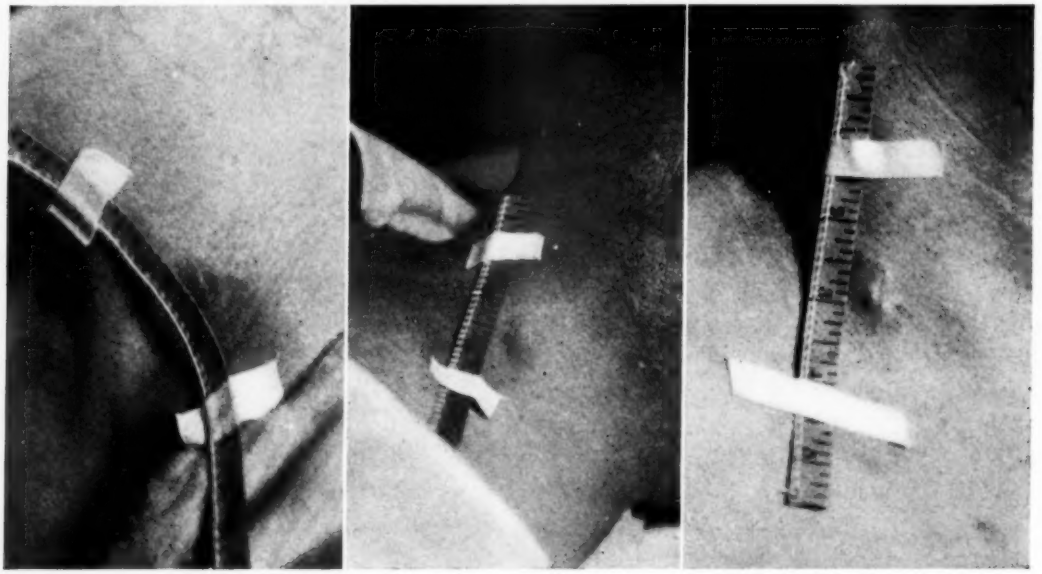
avulser is passed through the finger loop of an ordinary hemostat. The latter serves as a fulcrum as the avulser is being rotated. After the nerve is firmly clamped, it is cut with scissors as high above the avulser as possible. The nerve is then slowly twisted on the shank of the avulser as the handle is rotated. (Fig. 5.) The baffle at the distal end prevents the nerve from slipping off the instrument. It is important that the twisting motion be done very slowly. Rapid turning of the avulser may cause increased pain, and is very apt to cause breaking of the nerve by producing too much traction. The slower the nerve is dragged out of its deep position in the chest, the more likely is the entire nerve to be

obtained. Attachments of fascia to the nerve may interfere with avulsion, or may cause undue damage to surrounding tissues and should be freed from the nerve with a tissue forceps as the avulsion progresses. The avulsion should take at least two minutes, unless it is an especially easy nerve to remove. After a few inches have been removed there is the characteristic snap, and the nerve is free to be lifted out of the incision.

During the avulsion, it is well in all cases to allow the patient to have an analgesia produced by nitrous oxide and oxygen. This quiets the patient and prevents forced movements of respiration which might break the nerve prematurely.

There should be no hemorrhage after removal of the nerve. The tissues are sponged, and if dry, they are closed. A few interrupted sutures of number 0 plain catgut close the space in the fascia and platysma muscle. The skin may be closed with one skin clip which is removed on the third day. It has been our custom to place three clips close together, and remove two of them the day following operation, allowing the middle one to remain in place until the third day (Fig. 6). A folded piece of gauze is placed on the wound and held with two strips of adhesive.

The patient is returned to bed and given a regular diet unless specifically contraindicated. There are no routine postoperative orders for these cases. If a sedative is needed, codeine sulphate in half grain doses is given by mouth.



A B C

FIG. 9. A. Scar of this incision measures $\frac{3}{8}$ inch in length. Entire phrenic nerve which measured $15\frac{1}{2}$ inches was avulsed. At time of operation incision measured exactly $\frac{1}{2}$ inch. B. Scar in this case measures $\frac{3}{8}$ inch. At operation entire nerve measuring $13\frac{1}{2}$ inches was obtained. C. This photograph was made ten days after operation. It measured $\frac{7}{16}$ inch in length; $7\frac{3}{4}$ inches of phrenic nerve was obtained. These patients were all well nourished and no difficulty was encountered in removing nerve through a small incision.

COMBINED PHRENIC EXERESIS AND SCALENIOTOMY*

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IT is easily understood why many members of the medical profession are sceptical as to the value of surgery in the treatment of tuberculosis of the lung. Probably the most plausible explanation for this scepticism is that the surgical management of pulmonary tuberculosis itself is comparatively new. Until five or ten years ago many leading surgeons in this country felt that any attempt to compress the lung by a surgical procedure would soon be recorded in history as a passing fancy or another experiment that proved of little or no avail at the expense of unfortunate victims. Better knowledge of the value of these procedures has caused specialists, in more recent years, to refer for surgery many patients whose disease is far less advanced and whose better lung and other organs are more often wholly free of clinical tuberculosis. The progress in this field of endeavor is without doubt the most valuable contribution to the therapy of pulmonary tuberculosis since Detweiler advocated sanatorium treatment in 1870. In addition, those of us who have seen some of the mechanical and physiological results from many of these surgical procedures believe that there is, and will be, a growing demand for skilled surgeons to study and devise new methods of treatment both clinically and experimentally for this malady.

One must not get the impression from the foregoing that all cases of tuberculosis require surgery. On the contrary the majority of tuberculous patients are not suitable for surgical treatment. Operation is most commonly indicated when the lesions are chiefly unilateral, and in the comparatively few localities where modern surgical methods have been properly introduced and conducted there has developed the closest cooperation between the surgeon and chest specialist. It is therefore incumbent upon physicians everywhere to acquaint themselves with both indications and contraindications for surgery so that the thousands of patients that otherwise would be lost may be restored to an existence of comfort and usefulness.

Some years ago, it was demonstrated by Tuffier[1] and Jessen[2] that life could be sustained with a part of one lung. This was demonstrated by resection of the diseased portion. While this particular operation has since been abandoned it served as a stepping stone by proving that one could live with a part of the inherited lung tissue.

James Carson[3] pointed out as early as 1882 the value of compression in the treatment of pulmonary tuberculosis, and stressed the fact that the elasticity and mobility of the lung prevented in great measure its healing process in disease. He pointed out that the two sides of a lung abscess could not heal any more readily than the divided and separated ends of the tendo Achillis. In 1882 Forlanini[4] of Pavia first developed and practiced artificial pneumothorax and de Cérenville[5] of Lausanne in 1885, based upon his own work and also the work of Simon and Estlander in 1869 and 1879 respectively attempted to relax the tuberculous lung by a thoracoplastic operation. In 1888, Quincke[6] performed practically the same operation as advocated by Cérenville and in 1896 advocated small rib resections for both cavernous and non-cavernous types of tuberculosis. Carl Spengler in 1890 resected greater lengths[7] of the ribs and called the operation extrapleural thoracoplasty. During the first five years of the twentieth century, Turban,[8] Quincke,

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Garré[9] and others advocated thoracoplasty in tuberculosis and Bier[10] and Landerer[11] practiced the procedure for the treatment of the disease during the same period.

A B

Fig. 1. A. Mass of plaster inserted in cadaver chest cavity before scaleniotomy. B. Smaller plaster cast in same chest following scaleniotomy.

Their results from these less extensive resections were discouraging, but Brauer[12] realized that satisfactory improvements and cures could be obtained by additional collapse and immobilization to give more rest than the previous thoracoplasties afforded, and therefore proposed removal of the entire lengths of the second to ninth ribs inclusive. The mortality of this operation was so great that numerous changes had to be made in technique. Boiffin and Gourdet,[13] Wilms[14] and others worked for many years attempting to devise a more effective procedure to lower the mortality. Sauerbruch and Elving[15] in their 1913 monograph accepted the Wilms work of localization of the rib resections to the paravertebral region and pointed out the necessity of removing the first to the tenth ribs inclusive.

Phrenicotomy was first proposed by Sturtz in 1911 and by Sauerbruch in 1913.[16] It fell into disrepute however because of its failure to consistently paralyze the diaphragm, until it was revived by the work of Felix[17] and Goetze[18] in 1922, and Matson[19] and Plenke (1926) in Vienna. A confirmation of their work was made by us[20] in the University of Maryland School of Medicine in 1928–29. It has in more recent years been extended to a large variety of tuberculous conditions. We have practiced this operation for some years, recently resorting to the avulsive measure because the simple section of the nerve proved next to useless in the majority of cases. We have also felt the need of apical collapse associated with hemidiaphragmatic paralysis since the disease is most commonly located in or near the apex of the lung. A possible solution to this problem was suggested to the writer by Edward S. Welles of Saranac Lake, N. Y. in December, 1930. He had not performed the operation but had heard from some source that the scalenus anterior muscle could be cut with beneficial influence on a diseased apical process. I was unable to obtain additional

information relative to the procedure and upon my return to Baltimore began a study of its possibilities.

Fig. 2. Chest plate showing bilateral pulmonary involvement, more marked on right. This patient was given institutional care for over two years. His case was considered hopeless.

Fig. 3. Same chest as in Figure 2, two years later, with annular shadow in right upper lobe. Plate taken a short time before operation. Patient was having frequent pulmonary hemorrhages.

Physiologists' opinions differ relative to the function of the respiratory groups of muscles and I therefore decided to review the recent studies concerning them and to establish in my mind the function of each different group.

Since the time of Keith physiologists have taught that the diaphragm pushed down the stomach, liver, spleen and other abdominal viscera in a piston-like manner during inspiration and at the same time the costal margins were forced to diverge. Hoover[21] concluded after careful study of the functions of the respiratory muscles that the scaleni and intercostal groups are antagonistic to the action of the diaphragm. He concluded that the function of the diaphragm was first to increase and decrease the vertical diameters of the chest, and secondly to maintain the integrity of the costal margin. He also concluded that because the diaphragm is dome shaped its power to contract is lessened, giving the scaleni and intercostal muscles the advantage. This advantage causes the costal margins to diverge during the inspiratory period, the only role of the scaleni in this movement being immobilization or anchorage of the first ribs. The exception to the rule was found only under abnormal conditions such as pleural effusion, where there is no spasm of the diaphragm. In such instances the diaphragm loses its convexity and gains a mechanical advantage causing the costal margin on that side to converge instead of diverge during inspiration. These observations prove that there is antagonism between the diaphragm and the other groups of muscles both under normal and abnormal conditions. We have made similar observations both on dogs and man in cases of phrenic exeresis with unilateral diaphragmatic paralysis. Here the increasing dome of the diaphragm and the inability of the muscles to contract causes an accentuation of the lateral movements of the costal margin on the affected side during the inspiratory period.

The question then arose in our minds as to the part the intercostals actually play in respiratory movements. Galen believed that the external intercostals were

inspiratory and that the internal intercostals were expiratory. Vesalius, thirteen hundred years later, interpreted both

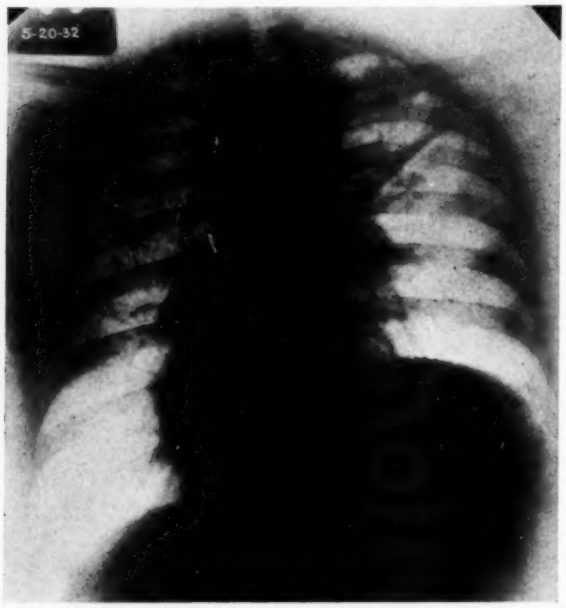

Fig. 4. Same chest six months after operation. Notice fibrosis at site of old lesion. Patient has now returned to work and appears to be entirely well.

external and internal intercostals as having the same function. Thomas Willis of Oxford in 1673 agreed with Galen that the external muscles took part in inspiratory and the internal in expiratory movements. In 1748 Hamburger devised a model to prove this. Hartwell and Martin isolated a pair of ribs by cutting away all intercostal structures which bound them above and below and left intact the blood and nerve supply to the space between the ribs. The external intercostal muscle uniting the two ribs was then severed throughout its entire extent and finally both ribs were cut so as to leave two segments that were connected only by the internal intercostal muscle. The blood and nerve supply to this muscle was still preserved. Artificial respirations were then employed and when automatic movements set up following cessation of the artificial movements the internal intercostal muscle was found to contract only during expiration.

Hoover[22] felt that this experiment did not prove that the external intercostals were never expiratory nor did it prove that the internal intercostals were never inspiratory. He, therefore, subsequent to a series of experiments, formulated the following thesis:

1. During tranquil breathing only the external intercostals are activated during inspiration and neither the external nor internal intercostals are activated during expiration.
2. During dyspnea the external intercostals are activated during inspiration, and in expiration only the internal intercostals may be activated, but when the abdominal muscles are brought into use either because of the resistance of the exit of air or by intense dyspnea then the external intercostals are synchronously employed with the abdominal muscles. The external intercostals are employed during expiration only when the abdominal muscles are requisitioned.
3. The internal intercostal muscles are employed during inspiration only in the presence of dyspnea or when there is an added resistance to the entrance of air.
4. The normal inspiratory and expiratory excursions of the ribs are determined not by the alternating action of the two sets of intercostal muscles but by the integration of these two sets of muscles with the muscles that convert the results of their action into an inspiratory or expiratory effect. The auxiliary muscles are the scaleni and the serratus posticus superior for inspiration and the triangularis sterni and the serratus posticus inferior for expiration.

With the foregoing theses in mind he further concluded that the scaleni muscles fix the first ribs so that the second, third and fourth ribs are rotated laterally and carried upwards so that there is an increase in the ventrodorsal and the transverse diameters of the thoracic cavity. To convince myself that the scaleni muscles only anchor the first ribs I stripped the soft tissues from the chest of a dog, exposed the ribs on the two sides and studied their movements both above where they join with the sternum at right angles and below where they join with it at a more acute angle. Except with extremely deep breathing I found the first ribs remained station-

ary. With the latter condition, however, the first ribs were slightly elevated during the inspiratory period. The second rises closer to the first, the third closer to the second and the fourth closer to the third. From the fifth rib down the interspaces remained about the same width both during inspiration and expiration. The scaleni muscles were then cut on one side and immediately the first, second and third ribs dropped downwards and inwards and failed to rise upwards and rotate outwards as they did prior to the severance of the scaleni muscles. There was a distinct asymmetry on the two sides. The scaleni muscles on the opposite side were then severed and the first four ribs behaved in a similar manner. The fourth ribs were almost stationary but the fifth and those below moved in their characteristic manner. Hoover[22] performed the same experiment on a dog some years ago and made similar observations. He felt that by virtue of the tone of the scaleni muscles the tendency of the first three ribs to descend is neutralized. It is our opinion, based on these findings, that after the first rib loses the support given it by the scaleni muscles and they descend and rotate inwards, they in turn serve as an anchorage for the ribs lower down. This stabilization enables the lower ribs to move naturally.

Since I was convinced that the first three ribs failed to rise during inspiration following scaleniotomy but rather contracted, narrowing the upper thoracic cavity, the question arose relative to the amount of collapse that would take place in the human chest following severance of these muscles. To determine this a cadaver was examined at autopsy a short time after death, the lung removed and the upper five ribs preserved. A mass of putty (Fig 1, A) was placed in the apical cavity, moulded in place and removed in toto. Subsequent to this a second amount (Fig. 1, B) was placed in the same cavity and the scaleni muscles severed. The results proved to us that there was considerable collapse but the putty was too soft to preserve the imprints of the ribs. We repeated the experiment on another cadaver, a short time after death, using French dental plaster instead of putty. There resulted a 40 per cent volume displacement in the second mass of plaster as compared with the first. Judging from these rough measurements I do not hesitate in estimating a 40 per cent collapse from the level of the third rib to the apex.

With the fact in mind that during the early stages of pulmonary tuberculosis the lesion is usually located in the apex of the lung, this procedure appeared to have a place in the treatment of such cases but not to be used independently. I did feel however, that scaleniotomy would be a distinct adjunct to phrenic exeresis, particularly in view of my experience with improvement of apical conditions with phrenic exeresis alone.

I was not unaware of the dangers that would be encountered in attempting to sever the scaleni muscles on a live human being. Our approach to the phrenic nerve for some time has been through a transverse incision, two fingers breadth above the level of the clavicle just behind the dorsal border of the sternocleidomastoid muscle. I felt, therefore, if it were possible to reach and sever the scaleni muscles, this incision would be ideal. The other practical route would be below the central tendon of the omohyoid muscle. Here the muscles in question could be isolated at their points of tendinous attachments, whereas, through the first mentioned incision they could not be isolated so easily. On the other hand, the higher site would be the better as with any attack below the level of the central tendon of the omohyoid muscle one necessarily encounters the transverse scapular and cervical arteries, the subclavian artery and vein, and in addition there would be grave danger of injuring the thoracic duct on the left side or the lymphatic duct on the right. With the incision for

exeresis of the phrenic nerve, the fibers of the scalenus anterior muscle can be picked from the ventral surface of the fifth, sixth, and seventh cervical nerves and severed below the level of the transverse process of the sixth cervical vertebra. This is necessary because some of the fibers of the muscle have their origin from the transverse process of this vertebra. When this has been accomplished the fifth cervical nerve can be retracted gently downwards and towards the midline. At this point it is to be remembered that the dorsal scapular nerve arises from the dorsal surface of the fifth cervical nerve trunk just as it emerges from the intervertebral foramen and pierces the scalenus medius muscle a short distance below its point of origin, after which it appears in the posterior triangle of the neck and passes downwards under cover of the levator scapulae and rhomboid muscles. In addition the long thoracic nerve arises from the fifth, sixth and seventh cervical nerve trunks as they emerge from their respective intervertebral foramina. This nerve likewise pierces the scalenus medius muscle at a little lower level than the dorsal scapular nerve, descending along the side of the neck behind the cords of the brachial plexus until it enters the axilla. In view of these facts the operative technique is as follows:

The patient is placed upon the table with the head resting on a soft pillow and turned towards the opposite side. An area extending from the lower jaw to the infraclavicular space and from the middle of the neck to some distance dorsal to the ventral border of the trapezius muscle is cleansed and draped in the usual manner. The landmarks, namely clavicle, midline of the neck, dorsal border of the sternomastoid muscle and the ventral border of the trapezius muscle are left uncovered. The level of the incision is determined by placing the fore and middle fingers just above the upper border of the clavicle. At this junction the scalenus anterior muscle is palpated and at this level 4 to 5 c.c. of ½ per cent solution of novocaine are injected in the cutaneous and subcutaneous tissue. The line of incision extends from a point about ¼ inch in front of the dorsal border of the sternomastoid muscle to a point just in front of the ventral border of the trapezius muscle, approximately 1½ inches in length. The knife is used only through skin, superficial fascia and platysma muscle and then Mayo dissecting scissors are used to uncover the ventral surface of the anterior scalenus muscle. Small blunt hook retractors are inserted and retracted in the line of incision. During the period of dissection until the scalenus anterior muscle is laid bare, one should depend upon the feel of the forefinger rather than upon sight to stay in the vicinity of the muscle. Once the muscle has been uncovered the phrenic nerve is located running downwards, inwards and slightly forwards across its ventral surface. The muscle and phrenic nerve are then injected with novocaine and the nerve is lifted out of its bed, dissected free, cut and slowly avulsed. Subsequent to the avulsion the scalenus anterior muscle is dissected free and a small periosteal elevator passed behind it, leaving the fifth, six and seventh cervical nerve trunks uninjured. All the fibers of this muscle are severed in this way except a few that come off from the transverse process of the sixth cervical vertebra. These fibers are picked up with rat tooth forceps and teased apart with scissors. The fifth cervical nerve trunk is gently retracted downwards and towards the midline. The scalenus medius and posterior may then be seen lying behind the cervical nerve roots. These are picked up and either cut or teased apart with blunt pointed scissors. Care is exercised during this latter step not to injure the dorsal scapular or long thoracic nerves already described. In addition we must be sure to sever the fibers that have their origin from the transverse process of the seventh cervical vertebra. This detail should not be overlooked because some

of the muscle fibers have their origin from the transverse process of this vertebra.

Throughout the entire operation one must proceed slowly and cautiously in order not to traumatize important structures. There is usually no bleeding except slight oozing from the severed muscle fibers.

With the combined phrenic exeresis and scaleniotomy the patient has scarcely more discomfort than with phrenic exeresis alone. The operation does not prevent him from being up the day following and he may be discharged from the surgical hospital on that day, but of course requires subsequent medical treatment, which is to be left in the hands of the chest clinician.

Judging from results obtained in my series of 70 cases since January, 1931, this combined operation has had a distinct advantage over phrenic exeresis alone. It is too early to pass final judgment on the procedure because clinicians feel that three to five years should elapse before passing final judgment, but the favorable response thus far obtained in a large percentage of the cases in this series, supported by the clinicians' reports, makes us feel that it is well worth a trial in cases of apical disease, both with and without cavity formation. We do not feel, however, that it should be employed where the disease is limited to the base of the lung. In addition we also believe the operation should be performed prior to all cases of extrapleural thoracoplasty.

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FEMORAL HERNIA

REVIEW OF THE OPERATIVE TECHNIQUE AND A NEW METHOD OF REPAIR

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WE have reached the age when we think our present-day operative technique is such that it cannot be improved upon. This being the case one hesitates to offer anything new.

On reviewing the literature of operations for the cure of femoral hernia, one is astonished to find so many methods and modifications in use.

Moschowitz reviewed the subject a number of years ago, and found seventy different methods. From that time the numbers have materially increased and it would be safe to estimate that a hundred or more exist today. When so many operations have been devised for a cure it means the results have been very unsatisfactory.

Having used my technique for over two years without a death or a recurrence, I think I have something as an improvement on previous operations for femoral hernia.

Operations for femoral hernia may be divided into two types:

(1) Low operation or old type.

(2) High or inguinal type, the later procedure.

The low operation is the older one, and by far the more often used even at the present day. The reason for this is that we have not gotten away from the old method of repair, probably because the high operation is the more difficult procedure. On the other hand the high operation gives a better percentage of permanent cures.

No operations for hernia are followed by so many recurrences as those performed for the cure of the femoral type.

The City of London Truss Society in a report covering the period 1921 to 1926 states that recurrence following the low type of operation was 27 per cent. This rate is surprisingly high.

In reviewing one finds that most surgeons have given their attention to the low type of operation. All seem to recognize that the obliteration of the femoral canal is essential for a successful operation. Many methods have been devised to accomplish this. McBurney after ligating the sac packed the canal with iodoform gauze. Macewen of Glasgow employed this sac as a plug. Pólya plugged the canal with the entire thickness of the sartorius muscle.

In the low operation not much more than simply removing the sac can be done. The complete removal of this is almost, if not quite impossible for anatomical reasons. After ligation and amputation of the sac there is a dimple or at least a small pocket at the upper end of the canal which is potentially the beginning of another hernia.

With the present-day operations, after the sac has been disposed of, the attempt is made to close the canal by sutures. One cannot expect that an approximation of two structures so tense as the inguinal ligament and the pectineal fascia or the ligament of Cooper can be permanently maintained after this type of low operation. After completion of these operations examinations show a weak and unsatisfactory closure.

Ochsner was one of the first to see this unsound surgical principle and the high percentage of failures following the attempts of plugging and sutures to obliterate the femoral canal. He advocated simple ligation and amputation of the sac. The principle underlying his method is to change the femoral canal into an unlined circular opening which will heal spontane-

ously. He reported a very low percentage of recurrences following his operation. Ochsner did not recommend his operation in the manner of dealing with the intestine and selection of the anesthetic. I believe as Coley and others do, in stressing the

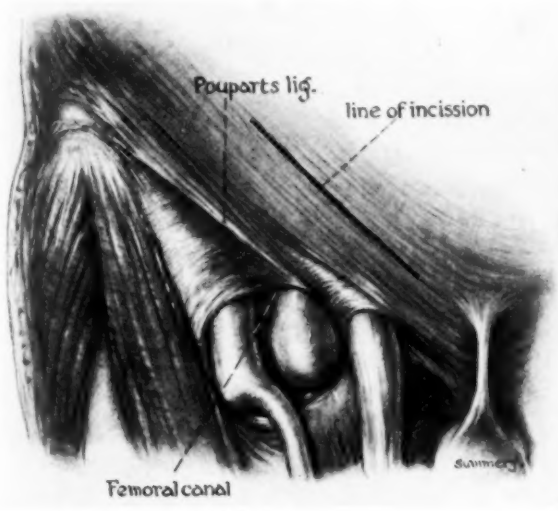

Fig. 1.

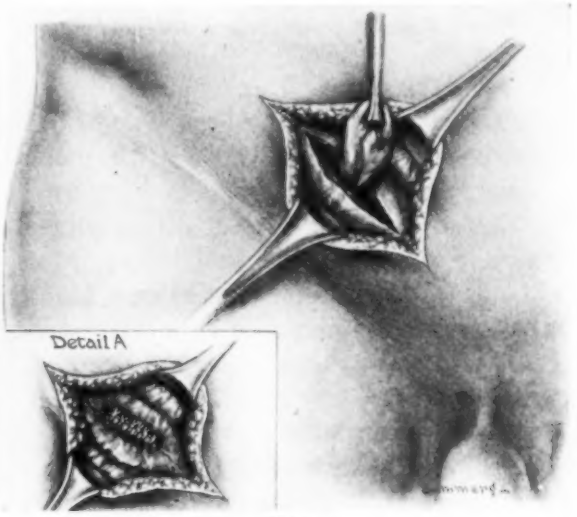

Fig. 2.

for strangulated hernias, in which the femoral ring had to be cut to permit reduction of the hernia. The weak point in this type of operation is a difficulty in ligating the sac high enough to avoid leaving a pocket, again there is no protection against recurrence until the canal becomes obliterated.

Moschowitz, Geoffrey Keynes and others hold that the low type operations should not be used. Much can be said in favor of the high operation as far as permanent recoveries are concerned. The usual type of high operation as recommended by Moschowitz is much more difficult than the low operation, and one cannot look too lightly upon the danger of injuring the bladder or femoral vessels.

I have seen the bladder opened twice, once by myself, and once by a colleague during this operation. The placing of sutures in a rather deep wound is not easy.

There was recently an article by Major P. Banerjee,[6] of Calcutta, India, advocating intraperitoneal herniorrhaphy in inguinal hernia.

My technique is the same with both simple and strangulated hernias, except essential importance of using a local anesthetic in the operation for strangulated hernias. If the hernia is not strangulated I consider the patient's choice of anesthetic. My procedure is as follows:

An incision is made through the skin 10–12 cm. long, parallel with and about 3 cm. above Poupart's ligament. It is practically the same incision that is used in the repair of inguinal hernia.

After the fascia is exposed the lower flap of skin is dissected downward until the hernial sac is completely isolated. The sac is then opened and the contents inspected, if it contains adherent omentum this is separated and reduced. I do not believe in removing a piece of omentum unless it is in bad condition from strangulation. If the sac contains intestine and the constriction is tight enough to prevent easy reduction, no attempt is made to reduce it until the constricting band is cut later on. The next step is to retract the upper flap and make an incision through the aponeurosis of the external oblique parallel to Poupart's ligament from 2–3 cm. above the inguinal canal. An opening is made through the internal oblique and transversalis muscles in a line parallel with

that of their fibers. After the peritoneum is exposed and an incision from 4–6 cm. long made in it a finger is hooked under the strangulated intestine. If gentle traction combined with slight pressure from below does not release the intestine, the constriction is cut from below. With the abdomen open and the upper end of the canal exposed one can easily see what one is doing when cutting the constrictions. After the constriction is cut and strangulation relieved, the injured intestine is lifted out of the abdominal cavity along with as much of the healthy gut as is necessary if resection is to be performed. With the intestine entirely freed and lifted out of the abdominal cavity the question of viability is more easily determined. If a resection is necessary, there is no danger of injury to the anastomosis as there is in reducing it in the low operation.

The next step is to free the hernial sac completely in the canal so that it can be inverted into the abdominal cavity. The sac is then picked up with the forceps and pulled up through the abdominal incision. If the sac is too large to be easily pushed up through the femoral canal, the distal portion is cut away. After the sac is drawn up through the abdominal incision the peritoneum is sutured all around it closing the abdominal cavity. The sac is then amputated flush with the fascia. It may be necessary to ligate one or two small vessels in the sac. The separated muscles are sutured together, the sutures passing through both walls of the sac, over this the fascia is then sutured with interrupted chromic catgut sutures with an occasional deep suture passing through the stump of the sac. The skin is closed in the usual manner without drainage unless there is an excess of adipose tissue or any danger of infection. If drainage is necessary a small rubber wick drain is introduced opposite the opening of the femoral canal and brought through the lower angle of the wound and is left in place for four or five days.

This is not a difficult operation and the surgeon has complete control of the situation.

By slight traction on the sac both walls of the upper end of the femoral canal are brought together, no pocket being left to invite recurrence. The thick walls of the sac give ample protection to the canal until it becomes obliterated after which further recurrence is impossible.

My reasons for recommending this operation are:

1. So far, I have had no deaths or recurrences. My belief is that it will give a lower percentage of recurrences than any other type of operation. Kocher had a similar way of using the sac in inguinal hernias of the indirect type. In 1914 I had the privilege of seeing him perform his operation for inguinal hernia, and heard him state that he claimed the best record for non-recurrence at that time.

2. There is no danger of injury to the bladder or femoral vessels.

3. It is simpler and easier to perform than any other type of high operation.

4. The intestine can always be inspected after a spontaneous reduction. There are many cases on record where the gangrenous intestine has slipped back before it could be inspected, followed by a fatal termination.

5. Intestinal resection is much more easily performed and there is no danger of injury to the anastomosis in reducing it.

6. The abdomen can be explored and the appendix removed. If there is trouble with the right adnexa it can be relieved. In a case where there happens to be an inguinal and a femoral hernia on the same side they both can be cured at the same operation. The question may come up in cases where there is a large femoral ring. "Will this type of operation hold?" In three of my cases it was necessary to cut the ring, making a larger opening than normal, but even in these there was no recurrence.

I wish to thank Doctor Patterson of New York who gave me the advice and courage to publish this method after seeing me demonstrate it.

[For References see p. 499.]

THE TREATMENT OF LAPAROTOMY INCISIONS*

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TO any patient who has undergone an abdominal operation, the skin wound and the healing process of it are of paramount importance. For this reason if for no other, the skin incision, the method of its closure and the subsequent treatment of the wound are worthy of a surgeon's careful consideration.

The method we describe is not new but has been tested and has been shown in practice to be successful.

THE "SKIN GRAFT INCISION"

This type of incision is made for any laparotomy. The wound is made by holding the knife at an angle of about 30° to the skin surface, using enough pressure so that the first sweep of the knife divides all the skin layers, exposing the subcutaneous tissues below. By holding the knife at this angle a graft is cut which is from one-quarter to one-third of an inch in width between the uppermost and lowest layers of the skin. The incision is deepened to the fascia beneath by holding the knife at right angles to the surface and without any departure from the usual routine.

CLOSING THE "SKIN GRAFT INCISION"

To close this incision a long strand of plain No. 0 catgut is used on a curved hand-sewing needle. The suture first closes the superficial fascia by a continuous stitch to eliminate dead space. In lower abdominal wounds this suture line unites the incision in Scarpa's fascia which can readily be recognized. After the suture has traversed the wound in the superficial fascial layer it is brought up in an angle of the wound just beneath the skin surface. From this point it is carried back across the wound in the opposite direction as a subcutaneous suture line. The last sweep of the needle brings the end of the suture up through the skin at a distance of about one inch from the end of the incision. It is fastened at this point by a slip knot so that just enough tension is maintained to keep the skin approximated. The bevelled edges now lie together but approximation is not accurate. By the use of "isinglass plasters" (a special type of the old-fashioned court-plaster as suggested by Dr. G. W. Roberts) an exact approximation of these bevelled skin edges can be made and held. The plasters are applied about one inch apart, an exposed space being left between them. The wound then shows only as a fine line where a break in continuity has occurred. The ends of the plasters are then moistened by a wet sponge, dried by pressure over a dry sponge, and fastened by the application of collodion. Care should be taken not to get the collodion onto the wound edges. A sterile towel is placed over the wound and the patient transported to bed.

OPEN METHOD OF TREATMENT OF WOUND

When the patient has been placed in bed, the bed clothing is folded down in order to expose the incision to the air. A blanket wrapped in a warmed sheet is placed over the upper portion of the body extending from the chin to the upper edge of the incision. Sterile towels are placed about the wound and the towel which has covered the wound up to this time is removed. In this way the so-called "double-bed" is prepared. Should there be any ooze

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between the plasters it is absorbed by blotting it with a piece of sterile gauze. When thoroughly dried (one to three hours) the bed clothes covering the chest and the sterile towels are removed. The gown is pulled down in immediate contact with the wound and the ordinary bed clothes are pulled up to cover the patient. No dressings are used at any time.

There are many advantages to this method of caring for the skin: (1) The most accurate skin approximation can be obtained. Common experience in other lines shows that it is much easier to bring two bevelled edges together with greater exactness than it is two square-cut edges. The sequel to this truth in this particular instance is that much less scar tissue is laid down in the process of repair. The final result in practically every instance is a thin white line which is gratifying both to the patient and to the surgeon. (2) There are no sutures to remove. Many patients dread "taking out the stitches" more than they do the actual operation. This knowledge is a source of great contentment to the patient. On the third or fourth postoperative day the ends of the isinglass plasters may be raised readily with the finger. As the patient turns in bed the plasters are gradually loosened and on the fifth or sixth day they drop off without the patient's knowledge. The end of the subcutaneous suture falls off as the catgut beneath the skin absorbs and breaks. (3) No dressings are used. This is of particular advantage to all concerned. The patients are happy in the knowledge that there will be no inconvenience or discomfort from repeated dressings. It also is a saving of money as the necessity for the use of dressing trays is eliminated. For the doctor it is a great time saver not to have to do dressings and wait for supplies. All he need do is to turn down the bed clothes. The wound can thus be visualized and consequently the healing process will be watched much more carefully. From the standpoint of the hospital many dollars are saved as the expense of preparing large quantities of dressing materials is greatly reduced.

DISCUSSION

In discussing this method of treatment with various members of the profession, many doubts as to the advisability of using the procedure have been raised. These are only natural and welcome. To every objection there is both a rational and practical answer. By answering the questions which have already been presented perhaps the doubts which have occurred to the reader may be eliminated.

(1) Will the superficial bevelled edge not slough? No. From the theoretical standpoint it will not occur because the blood supply of both lips has not been impaired. The process of healing therefore combines the ability of skin edges to unite and the probability of a homogeneous graft with good blood supply to "take." In practice, we have never seen such a slough occur.

(2) Is plain No. 0 catgut strong enough to hold? Let us first stop to consider why such materials as silk, dermol, horsehair, clips, etc. are used in the closure of the skin. In the first place, the security of the wound depends upon the approximation and healing of the deep structures, the peritoneum and fascia. The skin has no properties which prevent hernial development. Skin never has prevented the bursting of a wound or evisceration. Why, then, are heavy non-absorbable sutures ever used to close the skin? The object of skin-suturing is to approximate the edges and hold them in apposition long enough to allow healing to occur by first intention and to minimize the amount of scar tissue formed. With this in mind, it is apparent that the elimination of all needle punctures through the skin, together with the finest possible approximation of the free graft edges by isinglass plasters is the best method of accomplishing the result. In practice, we have never experienced the opening of a wound

because of too rapid absorption of the catgut.

(3) Does the wound become infected? The number of infections is very small as statistics prove. At the end of this paper we will present three sets of statistical figures which show exactly the wound morbidity with this method of treatment. However, there is behind this a definite rationale. Although we do not use any sterile surgical gauze for dressings, we do have a definite dressing for the wound which is much more efficacious. By exposing the wound to the air and allowing it to dry completely, there is formed over the fine line of the incision, a dried crust of serum. This is nature's most effective barrier against infection. Even though surgical dressings be applied, this natural barrier, rather than the gauze, is the real effective agent for preventing infection. When gauze dressings are applied the ooze is taken up by them and coagulates. The scab or clot thus formed embraces the network of the gauze. When this is removed the scab is torn from the wound. Nature's barrier to infection is thus inadvertently pulled off, adding materially to the possibility of infection. Our results bear out this fact.

(4) Can retention sutures be used? The presence or absence of such sutures makes no difference. The skin wound is treated in the same manner as described both in its closure and its after-care. No dressings are used. The retention sutures are tied loosely after the application of the skin plasters. The wound heals exactly as though no retention sutures were present. When these stitches are removed, the wound should be exposed to the air as it was immediately after operation, so that the stitch-holes in the skin are closed by drying of the serous ooze.

(5) What happens if a wound infection does occur? In this particular circumstance the absorption of catgut takes place very rapidly. Therefore, to open the incision and get proper drainage it is only necessary to make an opening through the skin with hemostatic forceps and spread the wound to any desired length with the gloved finger. By the time wound infections occur it will be noted that the skin healing has proceeded to such a point that it is separated with difficulty over uninfected areas and in these areas gaping never occurs.

(6) Is this method practical for both the private and staff patients? Between 1905 and 1915, Dr. G. W. Roberts did many operations in outlying districts and in private homes. The preoperative and postoperative care were carried out by the family physician. Over this period Dr. Roberts used this method with success. Since then it has been used both on the private and staff services at the Fifth Avenue Hospital, also with success. Any method of treatment which will withstand the severe test of use in a private home and postoperative care by a family physician can surely hold up under the care given in any modern hospital.

STATISTICAL STUDIES

In 1911 Dr. G. W. Roberts[1] first published a description of his technique in employing the skin graft incision, the "double bed," and the open method of the treatment of wounds. In 1927 Dr. G. W. Roberts and Dr. K. Roberts[2] reported a statistical study of the results of 112 cases treated by this method. In 1931 Dr. Ullyses Valdes[3] published a similar statistical study showing his results in using the open method of wound treatment in 110 cases. The present study covers a series of 265 cases in addition to those previously reported. Fortunately, Dr. Valdes used the same classification for cases as Dr. Roberts and we will continue the same way. This gives three separate series which can readily be compared.

Class A: Wounds in which there was no discharge of blood, serum, gas or pus during the healing process. A few drops of blood oozing from the wound before it is thoroughly dried immediately after

operation does not exclude it from this class.

Class B: Wounds in which there were no complications other than: (1) discharge of a small amount of serum in the absence of any sign of infection, lasting no more than seventy-two hours and in no way hindering the recovery of the patient; (2) the release of a small amount of air from an artificial opening made at one end of the incision; (3) the discharge of a small uninfected blood clot.

Class C: Wounds in which there was at some time a discharge of pus, the discharge however not lasting beyond the tenth postoperative day and in no way affecting the recovery of the patient.

Class D: Wounds in which any complication arose affecting the recovery of the patient.

This method of skin treatment was used in 265 laparotomies. The incisions used in these cases were: 16 upper right rectus, 111 modified Pfannenstiel, 92 McBurney, 23 Kocher, 20 inguinal, and 6 miscellaneous. Two hundred and thirty-nine of these incisions fall into Class A; 20 into Class B; 6 into Class C, and none into Class D.

TABLE I

Class	No. Cases	Per Cent
A	239	90.2
B	20	7.5
C	6	2.3
D	0	0

These figures are particularly interesting when compared with those obtained from an identically classified series done previously. The results reported by Dr. G. W. Roberts in 112 cases done during 1926, the results reported by Dr. U. Valdes done during 1930 and the present series are compared in Table II.

TABLE II

	Dr. G. W. Roberts		Dr. Valdes		Authors' Series	
No. Cases...	112		110		265	
	No. Cases	Per Cent	No. Cases	Per Cent	No. Cases	Per Cent
Class A.....	98	87.5	93	78	239	90.2
Class B.....	11	9.8	20	16	20	7.5
Class C.....	3	2.7	6	5	6	2.3
Class D.....	0	0	0	0	0	0

Let us strike the totals of these three series of cases. We then have a series of 487 cases which give the following results:

TABLE III

Class	No. Cases	Per Cent
Class A.....	430	88.3
Class B.....	51	10.4
Class C.....	15	3
Class D.....	0	0

We feel that these results are excellent and we therefore recommend this method of treatment of laparotomy incisions to the careful consideration of the surgical branch of the profession.

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INTUSSUSCEPTION

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FROM March 25, 1931 to December 2, 1931, 5 cases of intussusception due to tumor at the ileocecal region came under our observation. Four were in children and one was in an adult. In all, the tumors were benign. In four it was possible to remove the tumor at the primary operation. Two ablations were accomplished by intestinal resection and in the other two the tumor was excised or destroyed by the cautery.

Eliot and Corscaden,[1] reviewing 300 cases of intussusception in adults, 100 of which were due to tumors, state that tumors in children are of rare occurrence. In this small series encountered in less than one year, obviously this statement is not corroborated.

Of these cases the four children were males. In all but one the intussusception was found at operation. In Case v although intussusception was not found at laparotomy all the evidence points toward such a lesion which was spontaneously reduced, and in the reduction of which the anesthetic may have been a factor. In Case III, because of the poor condition of the child excision of the tumor could not be accomplished. Here an effort was made to prevent recurrence by fixation of the cecum and ileum.

"Polypoid tissue" or "glandular polyp" was the microscopic diagnosis of the tumor in these cases. Clinically it was believed that the tumor of Case I was malignant but this error was probably due in some measure to the distortion produced by the circulatory changes in the wall at that site attendant upon the intussusception.

CASE REPORTS

CASE I. M. M., aged twenty-seven, a white female, was admitted to the hospital March 25, 1931, complaining of cramp-like pains in the lower abdomen for the previous three days. These pains became worse on the day of admission and seemed to localize to the right side of the abdomen. There was no vomiting, belching, nausea or heartburn. Pain occurred periodically lasting from three to five minutes with intervals of relief from one to two hours. Enemata yielded normal stools with no blood present. Her temperature was 100°F., pulse 92, respirations 22, blood pressure 120/70. There was tenderness and some spasticity in the entire lower abdomen with an elongated mass palpable in the right lower quadrant lying transversely about 2 inches below the level of the umbilicus.

Operation: March 26, 1931; laparotomy under spinal anesthesia. There was an intussusception of the ileum into the cecum and extending upward through the colon as far as the sigmoid and an indurated mass with a broad base about 3 c.c. in diameter was palpated in the cecum at the margin of the ileocecal valve toward the colon after the intussusception was reduced manually. Because of the age of the patient, the induration of the mass and a family history of carcinoma, resection was decided on. The cecum with the appendix, a small portion of the ascending colon and a small portion of the terminal ileum were removed. An ileocolostomy (side-to-side) was done and the abdomen was closed. The postoperative course was uneventful. The wound healed by primary union.

Pathological Report: Polypoid tissue.

CASE II. J. C., four months old, male, was admitted to the hospital March 26, 1931. The baby was vomiting and crying at intervals. He became ill one day prior to admission when he began to vomit and have diarrhea. He cried at intervals of about every ten minutes, stopping and crying again as if he were having some sharp spasmodic pain. On the morning of admission the mother noticed there were streaks of blood in the baby's diaper. The baby had never been sick before. The abdomen was slightly distended and very tender. There was an elongated mass palpable transversely across the abdomen on about the level of the

umbilicus. Rectal examination revealed blood and mucus on the examining finger with the rectum ballooned out. Temperature 101°F., pulse 160, respirations 62.

Operation: Laparotomy revealed an intussusception of the ileum through the ileocecal valve into the colon and extending into the rectosigmoid junction. After the ileum was expressed from the colon manually, a sessile tumor about the size of a hazel nut was found at the upper portion of the ileocecal junction. A longitudinal incision was made through the cecum. The tumor mass was resected by means of the electrocautery and the cecum closed in three layers. The appendix was removed in course and the stump inverted.

Postoperatively the patient made an uneventful recovery.

Pathological Report: Glandular polyp with evidence of inflammation.

Case III. F. W., white male, six months of age, was admitted to the hospital April 25, 1931. The patient was vomiting, crying at intervals, and very restless. The mother stated that the baby became very restless that morning, refused its feeding and then began to vomit She gave the child an enema which resulted in a blood-tinged stool. The child had been delivered normally and never had been sick before. Temperature on admission was 99.2°F., pulse 116, respirations 56.

When the abdomen was palpated the child cried with pain. There was some distention present but no rigidity. A mass about 9 cm. long was palpable below and to the left of the umbilicus. Rectal examination revealed some blood on the examining finger. Soon after the examination was completed the patient passed a large amount of mucus and blood per rectum.

Operation: A diagnosis of intussusception was made and a laparotomy revealed the ileum intussuscepted through the ileocecal valve into and up to the middle of the transverse colon. The ileum was reduced manually. A small tumor about 8 mm. in diameter was palpable in the ileum about 1 inch from the ileocecal junction. The ileum was markedly edematous at this point. Because of the patient's poor condition no attempt at resection of this tumor mass was made but the cecum and terminal ileum were attached by a few interrupted sutures to the lateral pelvic wall to diminish the possibility of recurrence. The abdomen was closed in layers.

The *postoperative course* was uneventful.

Case IV. H. S., aged sixteen months, white male, was admitted to the hospital November 10, 1931. The patient was vomiting and crying as if in pain. The pain began that morning about six A.M. and was intermittent in character, about six or seven attacks of pain in an hour, each spasm of pain lasting only a few seconds. An enema revealed blood in the stool. The baby's health previously was always excellent. An older brother of this child had intussusception about four years previously and died apparently of the delay in making the diagnosis because at operation the intestine was already gangrenous. Temperature on admission was 100°F., pulse 150, respirations 48. The abdomen was distended and there was tenderness in the epigastrium and right upper quadrant. There was some spasticity of the right rectus muscle. In the region of the epigastrium a mass the size of a small apple was palpable.

Operation: A laparotomy was immediately performed under spinal anesthesia and an intussusception of the ileum up to the splenic flexure of the colon was found. The intussusception was reduced and a polyposis of the mucosa of the terminal ileum was found. About one foot of the terminal ileum containing the polyposis was resected to the ileocecal junction. A side-to-side anastomosis was done. The appendix was removed.

The *postoperative course* was uneventful except for an attack of tonsillitis lasting three days and a small break-down of the wound which granulated quickly.

Pathological Report: Polyposis of the ileum. Normal appendix.

Case V. M. K., male, aged ten, was admitted to the hospital December 2, 1931, complaining of severe pain in the right side of the abdomen, nausea and vomiting. The illness began that morning with severe intermittent pain in the right side of the abdomen. The patient was admitted to the hospital about twelve hours after the initial onset of pain. The abdomen showed tenderness and rigidity in the right lower quadrant. No masses were palpable. Pressure-release-tenderness was present over the right lower quadrant, but was not referred. Murphy's sign was negative. Rectal examination revealed tenderness toward the right side. The extremities and reflexes were normal.

Operation: Laparotomy under spinal anesthesia. The appendix was congested. The

terminal ileum was markedly congested, especially the last 2 inches, which were markedly thickened and inflamed as if they had been intussuscepted into the ileum and under the increased peristaltic activity of the intestines following the spinal anesthesia had been forcefully reduced. The mesenteric glands in the mesentery of the last loop of ileum were enlarged, inflamed and edematous. In these last 2 inches of ileum there was a distinct mass which seemed to have a flat base. An incision was made in the ileum about an inch and one-half from the cecal junction; a polypoid growth was found which was excised and the base, about 1.5 cm. in diameter, destroyed by means of the electrocautery. The appendix was removed. The patient made an uneventful recovery.

Pathological report of the tissue removed from the ileum was "glandular polyp with evidence of inflammation." The appendix was reported as having "no lesion."

ETIOLOGY

In 1884 in an exhaustive monograph by Treves[2] on intussusception it was stated that polypi were present in 5 per cent of all cases. Since then the publication of a great many more cases of intussusception resulting from polypi of the small intestine and tumors of the large intestine have resulted in the accumulation of sufficient statistics to enable their proper classification. Histologically these tumors range from simple polyps to lipomata, adenomata, fibroma, myxofibromata, myxomata, cyst of the ileocecal valve and papillomata in the benign group, and various varieties of carcinoma and sarcoma in the malignant group. In the small intestines the malignant tumors are about half as numerous as the benign whereas in the large intestine the proportion between benign and malignant is about equal. Obviously therefore the number of cases of intussusception in the small intestine resulting from tumors which are benign is much greater, because the percentage of benign growths is so much greater. This is also due to the fact that benign lesions produce less alteration in the spasticity of the bowel, the latter remaining much more pliable and soft.

D'Arcy Power[3] in a monographic review of the subject of intussusception in 1897 concluded that intussusception in children is almost always spontaneous in origin, whereas in adults far more frequently the cause is some form of new growth, although even then it may be purely spontaneous.

Eliot and Corscaden[1] reviewed and analyzed 300 cases of intussusception occurring in adults. In this series there were over 60 instances of benign tumor, the greater number of which were found in the small intestine, and 40 instances of malignancy associated with obstruction. Of the benign type the majority had their origin in the inner walls of the intestinal tube. Most of them had a well-defined pedicle from which the mass projected into the lumen, creating a condition far more favorable for the production of an intussusception than subserous tumors, of which type Willard[4] had reported a very interesting case caused by subperitoneal lipoma. King,[5] reviewing the statistics of the Mayo Clinic, found 7 solitary intestinal tumors out of 44,654 intraperitoneal operations; of these, 6 were lipomata and one was a myoma of the jejunum. Reviewing the literature he found 5 cases of adenomas of the small intestine, of the fibroadenoma and myxofibroma type. Of these mixed cases of fibroadenoma, 5 in number, only 2 were accompanied by intussusception. On the other hand Kassemeyer,[6] five years before the appearance of King's paper, collected the reports of 10 cases of adenoma of the small intestine complicated by intestinal invagination. Willard reported 3 cases of his own of benign adenoma of the small intestine complicated by intussusception and found in the literature 5 other cases of adenoma and intussusception not reported by either King or Kassemeyer. Since then there have been 2 other cases reported which were mentioned by Willard. This brings the total number of fibroadenomata of the small intestine complicated by intussusception to 20 cases.

Intussusception is said to be the cause of ileus in approximately 30 per cent of all cases. At least 2 per cent of cases occur in children under one year of age, yet it is sufficiently common in adults to merit close attention.

In a series of 59 cases recorded by McGregor[7] 4 were adults over 41. Codman[8] in 1908 stated that 27 cases of intussusception in the Massachusetts General Hospital during the ten years prior to 1908 showed 9 cases occurring in adults.

Of the 300 cited by Eliot and Corscaden in which there are 100 instances of tumors of the intestine, presumably responsible for the intussusception, they make the statement that tumor of the intestine as the cause of intussusception in children is very rarely encountered. The comparison of the exciting causes of the intussusception in children and in adults is very interesting. In infants where different kinds of acute intestinal disturbance resulting from disturbances in diet preponderate, pathological changes in the vermiform appendix contribute very slightly, if at all, to the development of intussusception. In younger children, however, Eliot and Corscaden have collected 18 instances in children between the years of two and seven in which an inversion of the appendix was probably the exciting cause of an intussusception. Only 3 such instances could be found in their collection of 300 adult cases.

In their adult cases they were able to collect 23 instances in which some form of trauma was associated with the intussusception. In regard to the various forms of trauma in these instances, those in which there was some form of violent muscular exercise predominate. In infants trauma occasioned by the muscular effort of crying and straining might increase the preexisting intussusception or predispose and be responsible for its development.

Ulcerations of the intestines, not associated with malignancy, were associated with intussusception in 19 of their cases in the small intestine and 13 instances in the ileocecal valve and additional 17 instances in the large bowel.

In 29 cases of Meckel's diverticulum associated with intussusception in both adults and children which they collected, 15 occurred in patients under ten; 5 between ten and twenty; 4 between twenty and thirty; and 4 in patients between thirty and forty-nine and in one the age was not recorded. They also direct attention to a considerable number of acute, subacute and chronic cases of intussusception in which no cause is mentionable or ascertainable.

Perrin and Lindsay[9] found the disease more common in males, in a ratio of 2.1 to 1. In their series 78.5 per cent of all the cases of intussusception were in patients under two years of age, while 69.5 per cent were under one year of age. Of these occurring within the first year, 72.7 per cent (which means 60 per cent of all cases of intussusception) occurred in patients between the ages of five and nine months. Brown,[10] who observed 31 cases, all of which were of the ileocecal variety, found 64.5 per cent occurring in the second half-year of life, nine occurring before this time and the remaining two over two years of age. Fifteen of the children were breast fed and one was bottle fed. Gray[11] reported 20 cases of which 15 were in males and 5 in females. All except three occurred under one year of age. Nine of the children were breast fed and nine bottle fed. Four of the latter group were originally breast fed.

The impression gleaned is that intussusception is of course primarily a disease of infancy occurring most frequently in well-nourished infants, usually breast fed. A review of the many factors claimed to be responsible for the production of intussusception suggest that single causes are probably never operative but that the mechanism is a complicated one consisting of numerous factors. Irritation of the mucous membrane undoubtedly had an influence on the production of the intussusception. In the history of the case immediately succeeding the onset

of the disease there often is either diarrhea or some error in diet.

Kock and Oerum[12] found that there was a peculiar geographical distribution in intussusception. According to them the English-speaking countries are more subject to the condition than either Germany or France. Their explanation for the greater frequency of the condition in both Denmark and Great Britain is that in those two countries castor oil is a favorite household remedy, and probably is an important factor in the production of the intussusception as a result of the violent and irregular peristalsis which it occasions.

Nothnagel[13] and Propping[14] produced intussusception in animals by stimulating a segment of the intestine with the faradic current. Such stimulation produced a contraction of the stimulated segment which became invaginated into the relaxed distal portion of the intestine.

Knapp[15] produced a similar condition by the injection of physostigmin. Such intussusceptions are only temporary however.

In a recent contribution on "The Involuntary Nervous System in Abdominal Disease" in which he generalizes regarding the distribution of sympathetic and parasympathetic nerves in their relation to intestinal function, Fraser[16,17] pointed out that the large intestine as far as the lower end of the pelvic colon is the site of a pure sympathetic distribution; both motor and inhibitory functions being distributed along this channel. He pointed out that most cases of intussusception occur at that point of the intestinal tract where the nerve control changes from a double supply consisting of sympathetic and parasympathetic fibers in the small intestine to the single nerve supply, the sympathetic fibers alone in the large bowel. At this point where the innervation changes, incoordination may occur. He believes that derangement of innervation is the cause of intussusception in early childhood (between the sixth month to the end of the second year) by producing an exaggeration of the normal function of the muscular coat, namely, peristalsis. He states that in the last 300 cases of intussusception operated on in the Edinburgh Children's Hospital, 295 had their beginnings in the small intestine, in the lower end of the ileum, where, after reduction, the original point of invagination remained apparent. He believes that during "rush" peristalsis, which is abnormally rapid peristalsis, and which may run the whole length of the small intestine, no harm can result as long as the wave of inhibition and relaxation precedes the contraction, which is the normal state of affairs in the small intestine, but that when the wave reaches the lower end of the ileum it encounters an area in which a new type of nerve supply is prevalent. Under certain conditions of nerve derangement the preceding wave of inhibition is not transmitted, with the result that the strong contraction of the peristaltic rush carries a circular area of gut into the distal segment as an invagination, thus starting the intussusception. In support of this idea is the finding by both Bayliss and Starling[18] that intussusception may be produced experimentally by paralyzing Auerbach's and Meissner's plexuses.

Meltzer and Auer[19] showed that the "rush" wave was a true peristaltic wave with a contraction preceded by a completely relaxed segment of intestine. They produced the phenomenon in rabbits by the simultaneous intravenous injection of the stimulant, ergot, and the depressant, calcium chloride. Fraser then suggested that perhaps some dietetic error in the child which combines the depressant and stimulant factor might be responsible for the development of "rush" peristalsis. The phenomenon was produced by Meltzer and Auer by several other drugs in combination but the two afore-mentioned were the most effective pair.

Intussusception is not likely to occur unless, accompanying such nerve distribution derangement, there is also some

structural peculiarity of the intestine or its attachment which would predispose to the disease. The presence of a mesentery to the cecum and ascending colon is constantly found when ileocecal or ileocolic intussusception appears, thus permitting of the necessary mobility of the bowel.

Leriche and Cavaillon[20] found a mobile cecum in 22 per cent of infants and only 17 per cent of older children and they maintain that the occurrence of an ileocecal invagination is possible only under such circumstances. Fraser considers another structural influence: the strong muscular intestine which is found in the well-developed child, in whom intussusception most frequently occurs, and he points out the rarity of the condition in the ill-nourished child with the atonic bowel.

The condition may be precipitated by abnormal peristalsis produced by the presence of pedunculated tumor and enlarged Peyer's patch or the inversion of a diverticulum.

Perrin and Lindsay[9] state that the examination of the ileocecal valve in a three-months-old infant shows it to be annular in shape and covered with masses of lymphoid tissue which project into the cecum for ¾ inch. The terminal inch of the mucosa of the ileum is also studded with masses of lymphoid tissue which form a complete ring around the lumen of the gut. In a child one year old the ileocecal valve is still very prominent, but the patch of lymphoid tissue in the terminal ileum, although well marked, is less developed. They maintain that the cases of intussusception occurring in the ileocecal region are due to abnormal amounts of lymphoid tissue, which in turn comes from a change from breast-milk to other foods producing gas or intestinal disturbances with associated lymphatic hyperplasia.

Lymphoid hyperplasia, a tumor or an inversion of a diverticulum apparently act in the same way in producing an intussusception. The mere increased weight of that particular portion of the intestines involved might be sufficient to cause its invagination into the more distally placed portion of gut. All three acting as a foreign body might be productive of hyperperistalsis. The foreign body reactions will precipitate either a dragging or a pulling of the portion of the gut in which the etiological factor is situated into the relaxed, dilated distal segment. In this connection it has been pointed out, however, that not infrequently the tumor is at some distance from the apex of the intussusception.

This could readily occur if there were a spasm of the gut around the site of the tumor with relaxation distal to it. The distal relaxation would easily allow the slipping of the constricted portion of the gut into it. This explanation was the one submitted by Perrin and Lindsay.

VARIETIES OF INTUSSUSCEPTION

The ordinary and normal intusussception, if it may be so called, is a downward one, that is the intussusception enters the outer or receiving layer, the intussuscipiens, from above and passes down toward the rectum. Ascending or retrograde intussusception has been reported, is rather rare and when encountered is almost always an antemortem phenomenon. There are four varieties of intussusception recognized: (1) The enteric, which is the invagination that occurs entirely within the small intestine. (2) The ileocecal, which is an exaggeration of the normal invagination present at the ileocecal valve. The apex of the intussusception is the ileocecal valve and the ileum is invaginated into the ascending colon at the expense of the latter. (3) The ileocolic; in this type the intussusception begins originally in the ileum, a short distance from the ileocecal valve. The apex of the intussusceptum passes through the ileocecal valve and the intussusception occurs largely at the expense of the ileum. (4) The colic intussusception, which consists, as the name implies, of an invagination of the colon within itself.

In an analysis of 321 cases of intussusception found in infants, Weiss[21] found that 24 per cent were of the enteric variety, 42 per cent of the ileocecal type, 10 per cent of the ileocolic and 24 per cent of the colic. In children he found 23 per cent enteric, 43 per cent ileocecal, 14 per cent ileocolic and 20 per cent colic. In adults 29.5 per cent were enteric, 34.5 per cent ileocecal, 9 per cent ileocolic and 27 per cent colic. Perrin and Lindsay in their analysis of 400 cases admitted to the London Hospital during a period of eighteen years found enteric in 67.5 per cent, ileocecal in 31.5 per cent, compound in 1 per cent, retrograde in 0.5 per cent.

PATHOLOGY

The local pathology in intussusception is considerably different from that in other cases of acute intestinal obstruction because of the mechanics involved. The mesenteric attachment of the invaginated bowel produces greater tension on the mesenteric side of the intussusceptum, which causes a curve with the convexity towards the mesentery, particularly marked at the apex. This produces a slit-like opening in the intestine, rather than a circular opening, the slit being directed towards the mesenteric side of the intussuscipiens. The bowel regards the intussusceptum as a foreign body which is violently stimulating its lining mucosa and is therefore constantly endeavoring to establish a ring contraction above it in order to squeeze it along the intestine. The effect of this contraction is exerted on the entering mesentery with the result that changes similar to those seen in a strangulated hernia begin to make their appearance in the invaginated bowel to which the prolapsed mesentery passes. The pressure first affects the thin-walled veins causing an acute venous stasis (the greater the congestion the greater the bulk of the intussusception) so that eventually the pressure at the intussusception neck is so great that the arterial supply is affected. The original changes beginning as edema and extravasation of the blood may soon pass into necrosis and gangrene. The swelling is most marked at the apex and along the convexity of the intussusception because these parts are free from pressure. The middle layer is as a rule more damaged than the central one. Inevitably following these changes there is an increased permeability of the intestinal wall to bacteria. They escape into the peritoneal cavity and a progressive peritonitis develops. The omentum becomes adherent over the surface and attempts to wall off a local perforation and prevents a terminal general peritonitis. Because of the pull on the mesentery and because of the constriction of the vessels which accompany it, gangrene in the intussusceptum is much more likely to occur than in the intussuscipiens and not only more likely to occur but to occur at a much earlier date. If it is set in, a sloughing of the invaginated portion with subsequent discharge through the rectum may, under exceptional conditions, occur.

SYMPTOMS

The symptoms of intussusception are different in the child and in the adult. In the child with few exceptions, the symptoms are similar to those resulting from intestinal obstruction, irrespective of the underlying condition. They are all referable to the sudden occlusion of the lumen of the intestinal tract. Most prominent is abdominal pain, colicky in character, accompanied by paleness of the child, drawing up of the legs, screams and usually vomiting.

Vickers[22] in an analysis of 834 cases occurring in children found this typical onset in 87.5 per cent of the cases. Failure to get the ordinary bowel movement is a very prominent symptom, instead of which there is very frequently the passage of blood and mucus. This is almost pathognomonic. There is a goodly proportion of the cases, however, in which this sign either fails to appear or appears very late so it should not be used as the diagnostic criterion. Special care should be exercised in arriving at the diagnosis of those cases

in which intussusception is suspected and which do not show the blood or mucus in the stool. In the analysis of Vickers cases 11 per cent showed no blood or mucus and in 17 per cent none appeared in the first hour. Of those cases in which the stool was positive for blood, in 20 it appeared only after the first twelve hours.

In most cases an abdominal tumor can be felt. The tumor itself is sausage-shaped, about 1 inch in diameter and of variable length, is hardened and becomes more definite during the spasm of pain, while it may almost disappear during the interval of freedom.

It may be detected anywhere in the colon which is accessible to palpation but most frequently is found in the left side of the abdomen. It very frequently is tender and manipulation produces spasm.

The blood which appears from the anus is usually bright red in color and usually intimately mixed with mucus, giving the red current-jelly appearance. In enteric intussusception, the ileocecal valve seems to arrest the outflowing of blood and this may occasion serious delay in the recognition and treatment of the lesion. Pure colic intussusception may persist for as long as six weeks without giving rise to acute intestinal obstructive symptoms, because of the wide lumen of the bowel and the adaptability of its mesentery, the two conspiring to prevent strangulation and incidently also prevent the appearance of blood in any amount.

DIAGNOSIS

In considering the diagnosis a history of severe intestinal colic occurring in a child or infant accompanied by the passage of blood-stained mucus from the bowel at once suggests the possibility of intussusception and the demonstration of a palpable tumor confirms the diagnosis, although the inability to palpate the tumor does not exclude the possibility. The absence of fecal matter from the blood-stained mucus is extremely suggestive. It must be differentiated from the acute enterocolitis. Both conditions affect infants and children; they are both frequently preceded by digestive disturbance such as diarrhea and vomiting; blood and mucus coming from the rectum accompanied by tenesmus are characteristic of both. Under these circumstances the crucial point in the diagnosis is the sign of complete intestinal obstruction. This can be ascertained in two ways; finding mucus on the napkin and bile in the blood. A very important point in differentiating the two is that in acute intussusception the onset is very sudden.

This condition must also be differentiated from Henoch's purpura which usually occurs in older children, six, seven or eight years of age, is never accompanied by true intestinal obstruction; always has fecal matter present in company with blood and mucus and instead of being able to feel a tumor by rectum, the rectal examination shows a characteristic edematous feeling of the mucosa and if there is an iliac tumor it is fixed, usually in the left iliac fossa and results from an infiltration of the colon with hemorrhage. Beside this, during the course of the disease ecchymotic spots make their appearance around the joints of the extremities.

When an intussusception protrudes from the anus, it may be mistaken for a prolapse of the rectum. The differentiation is made by the examining finger which in true prolapse cannot be inserted between the protruding tumor and the walls of the rectum, whereas in an intussusception the finger can be swept completely around the prolapse.

PROGNOSIS

The prognosis depends in a large measure upon the duration of the condition before recognition and treatment. The earlier the recognition and the earlier the treatment, the more likelihood of a cure.

The type of intussusception is also a factor; an ileocolic intussusception is the most difficult to reduce and a type which first shows gangrenous changes; enteric

intussusception comes next in these respects. The reducibility or irreducibility of the tumor is also dependent upon the time of existence of the intussusception. Spontaneous cure does not occur in more than 1 or 2 per cent and when it does occur it does so by spontaneous reduction, by spontaneous elimination of the intussusception or by fecal fistula forming above the intussusception and establishing a communication with the surface.

In cases of adenomyomata or fibroid adenomas being responsible for the onset of the intussusception there is much greater likelihood of the patient presenting all the signs of acute intestinal obstruction because there is correspondingly greater likelihood of a complete and early occlusion of the lumen of the bowel.

TREATMENT

The treatment of this condition is unquestionably surgical. Of historic interest is the treatment of this condition by the injection of air and water. In the air-reduction method a modification of bellows had been used to inflate the bowel through the rectum. Of course the inability to regulate the amount of air injected subjects the patient to the possibility of a perforation of the bowel. The injection of water is much more reliable and also much more successful. It is employed in the following manner: under deep general anesthesia a stream coming from a head pressure of about 2 feet is directed into the bowel through a long rubber catheter, the patient in the meanwhile being inverted so as to favor the inflow of the water. The inversion is best accomplished by a Trendelenburg position. The impact and pressure of the fluid upon the tumor aids and succeeds in reducing the obstruction in early cases. These methods are dangerous because of the early circulatory changes in the effected bowel which diminish the bowel resistance to pressure and favor perforation. Hipsley[23] recently recorded 100 consecutive cases treated by hydrostatic pressure with a mortality of 5 per cent and a complete reduction in 62 per cent. The statistics compare very favorably with those of Vickers,[22] who reports 75 cases of intussusception operated on in the Royal Alexander Hospital for Children, since 1918, with a mortality of 6 per cent.

The operative procedures vary according to the pathology encountered. After opening the abdomen the small intestine should not be allowed to present in the wound, the operative hand should be gently introduced and the intussusception located and mobilized into the wound. The tumor is then gently lifted up into the field of exploration and it is examined very carefully to determine the variety. The reduction is then sought for. Tension on the intussusception is usually ineffective and not without danger. The safest and most satisfactory procedure is to make backward pressure on the apex of the intussusceptum by successively enclosing more and more intussuscipiens in the hands in a retrograde direction. This, bit by bit, pushes the invaginated portion backward until the inversion is undone.

Where the reduction is difficult but apparently not impossible, the previous manoeuver may be augmented by pulling upon the entering loop. If this is not sufficient it is advisable to wrap the entire tumor in a warm wet pad and then with pressure applied equally in all directions, to squeeze the mass with the hands. This reduces the bulk by displacing some of the liquid from the wall into the lumen and then facilitates reduction by the previously unsuccessful measures. Tears in the serous coats of the bowel should subsequently be sutured.

The apex of the intussusceptum should then be carefully examined for tumors. Because of the edema from obstruction, there is often considerable difficulty in determining the presence of a neoplasm, and in such instances there should be no hesitancy in incising the intestine in a longitudinal direction to accurately ascertain the condition.

If a tumor is discovered it should be removed unless the patient's condition

warrants no further operative treatment. Whether this be a local excision or a radical removal by enterectomy depends on the judgment of the operator. In this connection it must be remembered that malignancies are far more frequently encountered in the large bowel and in the adult; that pedunculated tumors are far less likely to be malignant; that sessile tumors at the ileocecal junction in children are not necessarily malignant, but are frequently the result of benign glandular hyperplasia. Where the benignity of a sessile tumor is not questioned it should be remembered that (as described in Cases II and V herein reported) a complete destruction of the tumor without enterectomy may be accomplished by the cautery. A portion of the mass is first excised with the knife or scissors for histological examination and then the cautery destruction of the entire tumor down to the muscular wall can be accomplished. With the proper heat as shown by the dull cherry-red color of the cautery point, troublesome hemorrhage is eliminated. After removal of the neoplasm the longitudinal incision in the intestine is sutured in the usual manner. The value of this procedure lies not only in its ease of accomplishment but also in that it is a substitute for partial enterectomy with its much greater consequent mortality. Where radical removal by enterectomy is decided upon because of malignant potentialities of the neoplasm, it can be accomplished by any one of the standard methods best applicable to the individual case. In two of the instances herein reported, side-to-side anastomosis after closure of both ends was adopted.

It seems quite likely that in all resections in children the continuity of the intestinal canal will be most easily and safely restored by such lateral anastomosis after end closure.

In choosing an anesthetic, subarachnoid block deserves first consideration. It produces no shock, it prevents operative trauma by giving maximum relaxation, it makes reduction of the intussusception simpler, it produces no tissue irritation, it interrupts shocking impulses coming from the operative field toward the brain, it combats ileus by producing a relative vagus-augmentor preponderance on the intestinal tract, it does not abolish the cough reflex which is a factor in the production of aspiration-pulmonary complications, it is easy to produce both in adults and in children and it is safe.

Postoperatively all of these patients should receive sodium chloride 5 per cent solution intravenously to bring up the blood-chloride level. Fluids should be forced. If vomiting prevents oral administration, 20 per cent glucose intravenously accompanied by insulin subcutaneously and a hypodermoclysis is often a very valuable aid. Elsewhere we have shown that it does not combat acidosis by acting as a fuel, but that it has a decided value as a stimulant of the body defense mechanism through its effect on the reticuloendothelial system. Where reduction is impossible or where gangrene has developed, the condition of the patient determines the choice of procedure, which briefly outlined is either (1) some form of resection with intestinal anastomosis, (2) resection with an enterostomy, or (3) enterostomy after wrapping the intussusception in omentum, with subsequent resection and anastomosis after the urgent symptoms of obstruction have disappeared. The many individual procedures for any of these three plans need not be discussed here.

SUMMARY

1. Five cases of intussusception with intestinal tumors as etiological factors are reported.

2. All intussusceptions should be carefully examined at the apex for tumors and if the attendant edema is so great that it is confusing, there should be no hesitancy in incising the intestine to facilitate exploration.

3. All such tumors should be removed if the patient's condition does not prohibit it.

4. The cautery may be used very efficiently to remove benign sessile growths, thus eliminating enterectomy and its greater mortality.

5. The subject of intussusception is reviewed.

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THE CONTROL OF
INACCESSIBLE HEMORRHAGE

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IN January 1933 J. F. Baldwin[1] of Columbus, Ohio, reprints a paper which originally appeared under the title of "The Detmold Method of Controlling Inoperable Hemorrhage."[2] The procedure is that of firmly ligating the extremities at their junction with the body so as to impede the venous return of blood while not interfering seriously with the arterial flow. As Baldwin states the idea is an old one; indeed, it has been ascribed to that fountain-head of medical knowledge, Hippocrates.[3]

My first acquaintance with this form of hemostasis was in 1888 during my internship at Mount Sinai Hospital where it was known as the method of A. L. Loomis who, doubtless, learned it first hand from Detmold. Dawbarn[4] recommended ligation of the upper thighs as a preliminary to operations in which considerable loss of blood might be expected. I have applied it in this manner many times, especially in operations upon the lungs and to check concealed visceral hemorrhage such as bleeding from the gastrointestinal tract. It is also described in my book on Thoracic Surgery.[5] The method which I use is that of Dawbarn who employed elastic rubber bandages or, when these were not available, elastic rubber tubing, and in emergencies in the patient's home I have even made use of the rubber tubing of a fountain syringe. Constriction of the limb should also be employed in cerebral hemorrhage in the hope that its progress may be slowed or even checked.

The action, as Detmold has stated, is that of a depletion into the veins. In applying the ligatures it is essential that cyanosis of the extremities be produced, and it is well to assure oneself that the distal arteries continue to pulsate. The action of this procedure is, of course, the exact opposite to that employed for the purpose of checking bleeding in the distal part of an extremity as in accidental injuries when *complete* cessation of the circulation in the limb is necessary. Then, of course, both the arterial and the venous flow must be checked. In these circumstances swelling will not occur and the tourniquet may be left in place a long time. I have observed a case in which a rope tied around the arm for gunshot wound of the hand and forearm was kept in place for six hours without the slightest permanent injury to circulation or nerve function. When this kind of treatment is necessary the constricted part should be properly padded to avoid actual contusion.

In order to check inaccessible hemorrhage, however, swelling must occur and will to some extent be progressive so that the limb should not be constricted for more than an hour at a time.

In spite of the once frequent employment of the ligature to control remote hemorrhage the actual proof and measurement of the fall in blood pressure had never been recorded. In 1915 at the suggestion of Dr. Arthur J. Bendick of New York, I made a series of observations in normal individuals and demonstrated the action of the ligation on the systolic blood pressure before a Clinical Conference at Mount Sinai Hospital. I found that in adults the vascular tension could be reduced without harm by 40 or even 60 mm. Hg and that this reduction could be kept up for an hour provided there were no syncopal symptoms. My conclusion was that the thighs should be ligated one at a time and that when the ligatures were

removed the one first put on should be the first to be loosened.

When a sphygmomanometer is at hand the fall in tension may be noted as a guide to the condition of the patient. When the blood is once more returned to the general circulatory system it will be found that an hour will have elapsed before normal pressure is restored. Surgical measures directed to the source of the bleeding should be taken as soon as the ligatures are in place and if the operation has consumed more than an hour the ligatures must be removed.

When operating with the patient's blood pressure reduced by the segregation method meticulous attention must be given to hemostasis, with care for even the tiniest bleeding point for fear of recurrent hemorrhage when the blood pressure again reaches the normal. It should also be noted that segregated blood in the extremities has not been affected by the anesthetic since the ligatures should have been applied before the narcosis was initiated. Therefore, when the ligatures are removed the general circulation becomes diluted with blood unaffected by the anesthetizing drug and the patient's return to conciousness is correspondingly rapid.

Merely to illustrate the importance of continued watchfulness I will record the case of a young man who was prepared for a cranial operation. As a precaution I ordered preliminary ligation of the thighs but I did not personally witness the carrying out of the order. During the progress of the operation I was surprised and alarmed to note profuse bleeding for which I could not account. Many more vessels had to be secured than is usual in a case of this kind. To my consternation it was all accounted for when after the operation I asked that the ligatures be removed and I saw rubber bandages being carefully unwound from just behind the patient's toes up to the body! Fortunately there was final recovery.

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PERITONITIS

PATHOLOGIC PHYSIOLOGY INVOLVED IN TREATMENT

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A BETTER understanding of the abnormal physiologic processes that occur in peritonitis should lead to a clearer conception of the causes of symptoms and to better results in treatment.

ABSORPTION FROM PERITONEUM

Absorption of water, crystalloids, colloids and bacteria occurs through the lymphatic system and more particularly the blood vascular system of the peritoneum. Physiological processes of filtration, diffusion, osmosis, phagocytosis, and possibly some "specific" method of absorption as occurs in the intestinal mucosa during digestion, are involved in this process.[6,13,16] Peritoneal absorption is most pronounced during the first stages of peritonitis.[18,19] Apparently absorption is slight after the peritonitis has become well developed, unless the local defensive mechanisms are disturbed.

Active peristalsis, active digestion, moderately increased intra-abdominal pressure, retching and vomiting, active splanchnic hyperemia, increased osmotic tension of the blood, copious serous exudate and force of respiration increase absorption.[23] Splanchnic venous stasis, cold to the abdomen, well-developed plastic peritoneal exudate, intestinal inhibition and vasoconstriction of the peritoneal vessels lessen the absorption.

The first stages of intestinal inhibition must be considered a protective measure, at least until a well-developed plastic peritonitis has formed. Early attempts to overcome this inhibition by means of enemas, cathartics, hypertonic salt solution and so-called "intestinal stimulants" (especially eserine and pituitrin) prove deleterious in that they prevent attempts at localization. Hotz showed that animals with intestinal hypermotility and splanchnic hyperemia due to sectioned splanchnic (inhibitory) nerves succumbed rapidly to an experimental peritonitis.

The diaphragmatic peritoneum probably possesses no greater absorptive powers than the rest of the peritoneum.[19] Most lymphatics of the parietal peritoneum connect with those of the diaphragm, so that most of the lymphatic flow from the parietal peritoneum traverses the diaphragmatic channels before it enters the thoracic and right lymphatic ducts.[50]

PROTECTIVE FACTORS IN PERITONITIS

Often the absorption of bacteria is so active during the initial stages of peritonitis that the patient rapidly succumbs to an acute fulminating intoxication. It is possible but not probable that this absorption may be protective in nature, in that it represents an effort to rid the organism of bacteria while the local antibacterial forces are being marshalled into action. The local antibacterial forces are represented chiefly by cells possessing phagocytic properties. The peritoneal exudate possesses bactericidal powers. A well-formed peritoneal exudate apparently prevents the absorption of toxins introduced into the peritoneal cavity of the experimental animal.[18]

The ability of the peritoneum to form adhesions early is a protective mechanism in that these adhesions circumscribe the infection and often prevent it from becoming generalized. Years ago Yates called attention to the fact that a little food or a cathartic can transform a life-saving localization into a hopeless, spreading infection.

The importance of the omentum in circumscribing and limiting a spreading infection is too well known to require further comment.

FACTORS THAT CAUSE SYMPTOMS IN PERITONITIS

(1) *Bacteriemia and Toxemia.* Early in peritonitis, as was stated previously, there occurs an absorption of bacteria and most probably of bacterial toxins through the peritoneum. If the patient withstands this onslaught, then symptoms due to dehydration, disturbances of the acid-base balance, circulatory disturbances, distention, inhibition ileus, anoxemia and starvation manifest themselves. The work of Haden and Orr, Wangensteen, and many others indicates that no appreciable absorption of toxins occurs from the gut in non-strangulated cases of intestinal obstruction and that many of the symptoms of the so-called "toxemia" of intestinal obstruction are due to dehydration, demineralization and reflex disturbances of distention. If these deductions are accepted, then one must conclude that many of the symptoms of peritonitis with its inhibition ileus are due to similar causes.

(2) *Dehydration* occurs from vomiting and accumulation of excessive amounts of fluids in the intestinal tract. The distention of the small gut, besides causing an excessive accumulation of a fluid rich in NaCl, interferes with the re-absorption of these fluids. Loss of H_2O and electrolytes, particularly Cl ions, causes a disturbance of the acid-base balance, demineralization, disturbances of osmosis and diffusion, increased viscosity of the blood, impaired kidney function, loss of intestinal tone and anoxemia.[43] Many of these functions that are disturbed by the loss of H_2O and electrolytes play an important role in maintaining the bactericidal powers of the body.

(3) *Circulatory Disturbances.* There occur in peritonitis, after an early, transitory splanchnic vasoconstriction, a paralytic vasodilatation of the splanchanic basin and a compensatory decrease in the peripheral circulatory blood volume. As the case progresses, distention interferes with the venous return in the intestinal wall.* This stasis results in the production of a local anoxemia. This anoxemia produces an increase in local capillary permeability, resulting in the loss of plasma. McIver found that a fluid distention of the entire small intestine occurred when the animals breathed from the spirometer containing a low percentage of oxygen.[40]

As the peritonitic process advances we observe the development of a shock syndrome, with a decrease in effective blood volume, capillary stasis, cyanotic pallor and a drop in blood pressure.† It is possible that these circulatory changes are due in part to absorption into the blood of disintegration products of proteins, as Olivecrona, Cannon and others have attempted to show, or that they are due to adrenal cortex insufficiency, but it is also very likely on the basis of the evidence on hand, that the splanchnic vasomotor paralysis and venous stasis and the local loss of plasma play an important role in producing this shock syndrome.

(4) *Anoxemia* or excessive fall of the diffusion pressure of oxygen in the systemic capillaries occurs, particularly in the more advanced stages of peritonitis, and is due to a combination of factors, the most important being: (a) Slowing of the circulation due to hemoconcentration and increased viscosity of the blood, conditions which result from plasma loss. Under these conditions an excessive proportion of oxygen is used up in the capillaries. (b) Alteration of the dissociation curve of oxyhemoglobin so that it gives off oxygen less readily (Bohr effect).

* That the splanchnic venous return is impaired is readily understood when one recalls that normally the venous return from the abdominal cavity is due principally to the head of pressure on the arterial side, and to a lesser extent to the pumping action of the contracting gut and diaphragm, to the respiratory excursions, to the venoconstriction in the portal system and to the venopressor mechanism which is dependent upon good general muscular tonus. In the more advanced stages of peritonitis these various functions that aid in the venous return are markedly disturbed.

† As a result of hemoconcentration from local loss of plasma and because of impaired venous return, insufficient blood reaches the left side of the heart to maintain arterial blood pressure.

This can occur when an alkalosis results from vomiting and from the accumulation of excessive secretions in the gut. (c) A pronounced lessening of vital capacity occurs as the result of interference of diaphragm action, as well as from interference with abdominal and lower costal respiration by the reflex abdominal rigidity and pain. (d) Rapid, shallow breathing can result from abdominal distention, fever and the depressing action of "toxins" upon the respiratory center. (e) Patients with peritonitis are predisposed to atelectasis because of lessened vital capacity and shallow breathing. (f) Cardiac function is embarrassed by hemoconcentration, demineralization, "toxemia" and impaired venous return, thus adding to the slowing of the circulation. (g) Deep morphinization, recommended by some in the treatment of peritonitis, produces pulmonary subventilation. (h) Certain microorganisms apparently can incite the conversion of some of the hemoglobin to methemoglobin.

When the respiratory center suffers from anoxemia, the respirations become shallow and rapid, resulting in improper aeration and aggravation of the anoxemia.

The defensive mechanisms of the body against bacterial toxins are lowered by anoxemia. Anoxemia and deficiency of CO_2 from rapid breathing may cause failure of the heart muscle.[33]

(5) *Distention.* The distention of the gut increases the amount of secretion into the intestinal tract. The re-absorption of these excessive secretions is interfered with in direct proportion to the degree of distention.

Gases collect to excess in peritonitis, due to swallowed air and to fermentation in the non-motile gut. Elimination of intestinal gas occurs by peristaltic activity and by diffusion into the portal circulation.

Absorption of intestinal gases into the portal system is ordinarily quite considerable, and any disturbance of the intestinal circulation as occurs in peritonitis interferes markedly with this process.

Atmospheric air that enters the stomach by swallowing as the result of nausea, belching, retching, vomiting, dry throat and anesthetization is resorbed into the blood stream with difficulty, and because of inhibited intestinal motility it is moved onward with difficulty.[40,41,57]

That it is the distention of the bowel that causes interference with the local circulation and thus influences the muscular activity of the bowel, is indicated by the experiments of Hotz.[34] He showed that loops of bowel covered with a thick purulent exudate would still manifest some activity, but once the gut became quite distended the intestinal movements were completely inhibited.

Distention of the gut produces reflex symptoms. These reflexes are responsible for such symptoms as anorexia, nausea and general depression. According to both Meek and Ivy, these symptoms are conspicuously absent when the distended loop had previously been de-nervated.[66]

Frequently in the earlier stages of peritonitis, distention only of the small gut occurs. This observation substantiates the assertion that enemas are often not only useless but harmful.

(6) *Inhibition Ileus.* In peritonitis the inhibition of intestinal movements is due to a large extent to an increased inhibitory action of the splanchnic nerves. Experimentally it has been shown that marked irritation of the parietal peritoneum can reflexly diminish the tone and contractions of the small gut and that the splanchnic nerves are the efferent paths for these reflexes.[36]

The splanchnics also contain vasoconstrictor fibers, which are paralyzed after a brief early period of stimulation in peritonitis. Since intestinal movements are dependent upon the blood flow, the circulatory disturbances that occur as the result of splanchnic vasoconstrictor paralysis and distention undoubtedly aggravate the ileus that results from reflex splanchnic nerve inhibition. The lessening in intestinal tone that results from hypochloremia is another factor.

That the splanchnic nerves play an important role in the production of the "paralytic" ileus of peritonitis is shown by Arai.[2,3] Peritonitis had little effect on peristaltic activity when he cut the splanchnic nerves prior to the production of the peritonitis. Undoubtedly the failure of response to nervous excitation in these cases is due not to paralysis of the conducting mechanism, but to reflex splanchnic inhibition.

The peritoneal irritation that induces excessive splanchnic inhibition also results in the production of excessive tonicity of the ileocecal sphincter. This increased tone of the sphincter may be a factor in causing gas to be "trapped" in the small bowel.

INCREASED INTRA-ABDOMINAL TENSION

This is due to distention of the gut, reflex abdominal rigidity* and excessive accumulation of fluid exudate in the peritoneal cavity. The first two are the most important factors in most cases.

The distention of the gut interferes with the function of the diaphragm and the reflex abdominal rigidity interferes with the respiratory function of the abdominal and lower intercostal muscles.

VOMITING

The early vomiting of peritonitis is due to irritation of the visceral peritoneum. In the later stages the vomiting becomes regurgitant in type due to the excessive accumulation of fluids.

DISTURBED INTESTINAL GRADIENT OF FORCES

Normally strength of intestinal contractions, tonus, rhythmicity and irritability are most pronounced in the duodenum and gradually diminish as the colon is reached. In peritonitis, as the result of splanchnic inhibition this gradient of forces is considerably flattened. Irritating enemas, pituitrin, ether anesthesia and many solutions given by protoclysis flatten the gradient and are to be avoided. Solutions when given by protoclysis are frequently unabsorbed, and by filling the colon they augment the abdominal tension and distress the patient.

THE LIVER AND PERITONITIS

It is assumed that absorption of bacterial toxins occurs through the peritoneum in cases of peritonitis, and if it does, then some of these toxic substances pass through the liver, especially when the omentum and visceral peritoneum are involved. This, as well as the disturbances of the splanchnic venous system that occur as the result of peritoneal inflammation and gut distention, indicates that the liver plays an important role in peritonitis. One must not forget that a liver well filled with glycogen is better able to perform some of its functions and that starvation, anesthesia and large doses of morphine deplete the liver glycogen store.

HEART AND PERITONITIS

That myocarditis (myocardial failure) does not commonly complicate the usually favorable cases of peritonitis is probably not sufficiently recognized. A diagnosis of myocarditis is too often made because these patients have a rapid, small pulse and because they often display a dusky suffusion. The rapid pulse can be explained as being due to an effort to compensate for a diminishing venous return to the heart as well as being due to the reaction of increased body temperature. The lowered blood pressure can be explained as being due to hemoconcentration and capillary dilatation of anoxemia.

That bacterial toxins can affect the myocardium cannot be denied, but in the non-lethal cases that have withstood

* Since the rigidity of the abdominal wall is dependent upon parietal and visceral irritation involving a neuromuscular reflex and since the response of this reflex is influenced by such factors as age of patient, state of nervous system, fatigue of the reflex, toxic poisoning of the abdominal muscles, etc., it can be appreciated that little or no rigidity occasionally may occur with a severe peritonitis.

the early bacteriemia, these other conditions that produce symptoms similar to a "myocarditis" must be given serious consideration.

ADRENAL CORTEX AND PERITONITIS

Swingle and his co-workers have demonstrated that by suppressing the adrenal cortex hormone they were able to reproduce the characteristic symptoms of secondary shock. Since in the more advanced stages of peritonitis one finds circulatory changes simulating secondary shock, it is not at all improbable that adrenal insufficiency is a factor and that injections of adrenal cortex hormone will be of value in drawing fluid back into the blood stream through the capillary walls in these cases and thus aid in restoring blood volume, blood constituents and blood pressure.

POSTOPERATIVE PERITONITIS

Most cases of postoperative ileus that occur within two to four days after a laparotomy, are due to a local peritonitis and the obstruction is due usually to matting of several loops of bowel by a fibrinous exudate, associated with excessive splanchnic inhibition. Cases of postoperative ileus that occur from the fifth to the tenth day are usually due to a localized peritonitis that has resulted in abscess formation, and frequently in these cases there occurs a mechanical obstruction due to kinking of a loop of bowel.

Peritonitis is the cause of death in 80 per cent of the fatal cases of appendicitis.[59]

SOME GENERAL PROBLEMS OF TREATMENT

Although progress is being made in the use of vaccines, bacteriophages and sera in the treatment of peritonitis, we must at present rely principally upon surgical and supportive measures.[37]

Unfortunately most cases of peritonitis are secondary to an intra-abdominal lesion and are usually due to infection by more than one organism. According to Melleney these polymicrobic infections are more severe than infections by a single organism.[44] Steinberg has demonstrated that the intravenous administration of anticolic serum prevents death in rabbits injected intraperitoneally with lethal doses of B. coli.[60–62] Scarlatina antitoxin is apparently of some benefit in some cases of peritonitis due to a streptococcus infection.

The sensitivity to infection, intoxication, anesthetization and trauma of operation is increased by dehydration, demineralization, slowing of circulation, hemoconcentration, anoxemia, disturbance of acid-base balance and depletion of liver glycogen. Fleming has shown that slight lowering of the NaCl content of the blood causes a great fall in phagocytic activity.[24]

The organisms most commonly found in the peritoneal exudate are B. coli, streptococcus and B. welchii. As stated in a previous paragraph, peritoneal absorption apparently diminishes as the peritonitis becomes fully formed.

After exudation and localization have occurred, it is the deleterious effects of distention and circulatory changes that usually preponderate. It is a fact that if a patient is able to overcome the early bacterial intoxication then the re-establishment of intestinal motility and correction of the circulatory disturbances remove the main dangers of a fatal termination later. In the early stages, absolute rest of the individual as a whole, as well as lessened respiratory effort and rest of the digestive tract, are essential in preventing absorption and in stimulating efforts at localization.

REMOVAL OF PRIMARY FOCUS OF INFECTION

Since peritonitis usually starts from some focus of infection in the peritoneal cavity, it behoves us to remove as early as possible this focus which serves as a source of further contamination.* If this

* An exception of this rule would be the pelvic peritonitis due to puerperal sepsis, because these cases do best when the primary focus is left alone. The

focus can be removed before the local defensive mechanisms have started, then no harm can come from the performance of a laparotomy, but once this local mechanism has started to organize then harm can result from the performance of an operation that entails the breaking-up of this mechanism. This is the basis of the so-called "Ochsner treatment" of neglected appendicitis. There occur cases, however, of so-called neglected appendicitis that do not "localize" and I refer particularly to the acute appendicitis of children. There is a small group of delayed appendicitis cases in adults that shows no tendency towards localization.[35] The mortality in these cases is very high when treated conservatively and it is probably just as high following an appendectomy. Extraperitonealization by means of incision of the peritoneum and drainage down to the appendix, without attempting its removal, performed under local anesthesia, is considered by some surgeons to be the best procedure.

In a recent personal communication Dr. Frederick A. Coller had this to say about his experience with the treatment of delayed appendicitis: "Our mortality in a group of patients who had had appendicitis beyond forty-eight hours and who had a general peritonitis was entirely too high and I decided to go back to the treatment advocated by Ochsner. Our mortality dropped from 50 per cent to 6 per cent in this group."

It is probably a surgical fact that the average delayed case of appendicitis that does not appear to be spreading would do better after immediate appendectomy if we all possessed the surgical skill that would enable one to remove the focus of infection without disturbing the defensive mechanism. However, a modified Ochsner treatment is the safest procedure, especially since most of these cases are handled by the occasional operator and general practitioner.

infection, being localized in the true pelvis, is very readily circumscribed, and the removal of the primary focus, entails entering the healthy peritoneal cavity and breaking through the wall of defense.

TREATMENT OF ANHYDREMIA

Since a normal, resting individual, whose fluid intake has been cut off, loses at room temperature about 2500 c.c. of H_2O daily, it is essential that this amount be replaced, in addition to the amount a patient with peritonitis loses through vomiting, accumulation of excessive secretions in the distended gut and from excessive perspiration.[56] This loss of H_2O and electrolytes represents a considerable amount. If one uses the blood-chloride determinations, urinary output and thirst as guides, the average peritonitis case will often require amounts in excess of 4000 c.c. daily, especially during the early stages.[48]

Ringer's and Hartmann's solutions are probably preferable to normal salt solution. The calcium ion probably aids in decreasing capillary permeability and the K, Na and Ca ions in the proportions contained in these solutions play an important role in controlling the rhythmic activity of the heart. The calcium, furthermore, displays a stimulating action on phagocytosis.[1] The calcium ion is also probably of value in aiding the repair of liver damage that might occur in peritonitis.

TREATMENT OF CIRCULATORY DISTURBANCES

In severe peritonitis with marked circulatory changes, little benefit is gained from normal saline, Ringer's or Hartmann's solutions, because these solutions "leak" out of the dilated capillaries very quickly. Six per cent acacia solution by fulfilling the function of plasma proteins raises the colloid osmotic tension and should prevent this leakage. Hypertonic glucose and saline solutions should not be added to acacia, because by attracting water from the tissues they dilute the plasma and lower the colloid osmotic tension.[15,63]

A good combination for these grave cases is one containing acacia and minute amounts of pitressin to stimulate capillary

tone. (It is not unlikely that injections of adrenal cortex hormone will aid in restoring blood volume and blood pressure.)

USE OF GLUCOSE

Glucose is definitely indicated, especially in the later stages, to overcome the effects of starvation and to help maintain basal heat production. Because glucose exerts diuretic properties, it causes the loss of electrolytes from the body. For this reason it should be given in normal saline solution.

CONTINUOUS VENOCLYSIS

The Ringer's, glucose and acacia solutions are best administered by continuous venoclysis. According to Hirschfield, many reactions that follow intravenous injections are due to the speed with which the solution is introduced ("speed shock"[32]).

Continuous administration of normal saline and glucose solutions, when anhydremia does not exist, can result in a dilution of the blood plasma. This results in a reduction of the colloid osmotic tension of the plasma and fluid escapes from the plasma into the tissues, where it is not needed.

Use of the positive-pressure bottle as devised by Mulholland of the Mayo Clinic is the simplest way of administering fluids by continuous venoclysis.

Proctoclysis. The only solutions that are ordinarily readily absorbed by means of proctoclysis appear to be H_2O and isotonic salt solution, and even these are frequently not absorbed by the cecum and ascending colon. If withholding everything by mouth is so important in producing physiological rest to the bowel, then why should we not withhold solutions given per rectum, particularly in the early cases of peritonitis due to a gangrenous appendicitis?

FOWLER'S POSITION

This position, when originally advocated, was based upon experimental misconceptions. Nevertheless, in early cases of peritonitis it possesses distinct advantages, in that gravity permits the localization of infective matter in the lower portion of the peritoneal cavity, where it can be more easily circumscribed. It is this tendency of fluid to gravitate that accounts for the fact that most cases of peritonitis start in the pelvis, even those that result from perforating lesions high up.

The semirecumbent posture, even though it favors gravitation of fluid toward the pelvis in the early stages has little effect as far as peritoneal absorption is concerned. Fowler's position increases the ease of respiration in distended patients by the weight of the liver assisting the contractions of the diaphragm.* Posture no longer influences the gravitation of fluid once the intestines have become distended and glued together by an exudate.

Fowler's position when continued over a number of days is apt to cause interference with the venous return from the pelvis and thus predisposes the patient to phlebitis and the dangers of pulmonary embolism.

In the more advanced peritonitis the sitting position sometimes can do harm. Marked diminution in volume-flow usually exists in these cases, and the heart and vital centers in the brain are liable to suffer early. One must not forget the deleterious effect of the upright position upon the brain when the blood pressure approaches the critical level (80 mm. Hg).

If these deductions are correct, then one must recognize the fact that Fowler's position is of especial value in the earliest stages of peritonitis. Later the comfort of the patient should be the most important determining factor as to the best position he should be placed in.

The brilliant results that follow incision per rectum of appendiceal abscesses that point into the rectum, suggest that gravity plays an important role in obtaining these results. Turning the patient upon his side following the incision of an intraperitoneal

* It is an experimental fact that restriction of the depth of breathing produces anoxemia much more easily in the recumbent position than the sitting position.

abscess will often favor drainage by gravity of a localized exudate.

OXYGEN THERAPY IN ADVANCED CASES

That anoxemia can result in peritonitis is recognized, but that this anoxemia can be favorably influenced by the early institution of oxygen inhalations is probably not sufficiently recognized. Inhalations of oxygen can increase the amount of oxygen in combination with the hemoglobin, when it is not being saturated, and they can increase the amount of oxygen that goes into solution in the plasma. Since oxyhemoglobin in a normal, resting individual parts with about 5.5 c.c. of oxygen to the tissues for every 100 c.c. of blood, the increased amount of oxygen that goes into solution in the plasma as a result of oxygen treatment probably represents a not inconsiderable portion of the easily available oxygen to the tissues.[28–30]

These estimates show the physiological importance of oxygen inhalation when a state of anoxemia exists. However, diminished venous volume return, capillary stasis, diminution of effective blood volume and plasma loss should be compensated for in order to get any real benefit from this increase in oxygen.[58]

The improved oxygenation of the blood in infections stimulates the immune reactions, particularly phagocytosis.[1]

Since CO_2 rebreathing acts as a stimulus to the respiratory center, it might be well, especially when the respirations are shallow, to combine low percentages of CO_2 with the oxygen inhalations. CO_2 rebreathing causes more complete expansion of the lung by increasing depth and amplitude, thus expanding atelectatic areas. CO_2 however causes a dilatation of the capillaries probably because of its acidity and must be used cautiously.

THEORETICAL BENEFITS OF EPHEDRINE IN EARLY CASES

Small doses of ephedrine (1/8 gr.) should be able to overcome, in the earlier stages, the effect of paralysis of the splanchnic vasomotor nerves. Its action is not upon the vasoconstrictor fibers, but upon their myoneural junctions. It produces some inhibitory effect upon gut motility, as well as improvement in the intestinal circulation due to vasoconstriction and increased cardiac output. Its inhibitory effect upon gut motility is of advantage in the early stages in that it aids attempts at localization and lessens absorption.

Since in the early stages of peritonitis, absorption is diminished by producing vasoconstriction of the splanchnic vessels, it would appear that ephedrine, because of its vasoconstricting properties, would be advantageous.[22] Furthermore, its vasoconstricting properties should lessen the amount of "weeping" from the peritoneum, as well as lessen the amount of plasma loss due to gut distention and local anoxemia.

The effectiveness of ephedrine is interfered with when the circulation is markedly impaired. Ephedrine is contraindicated in advanced cases of peritonitis with circulatory damage, because it exerts a depressant effect upon the heart under those conditions.[9,10]

USE OF MORPHINE

If it is true that inhibition of intestinal movements is a protective measure in the early stages of peritonitis, then any beneficial effect that can come from the liberal use of morphine in this stage cannot very easily be ascribed to its action upon the small intestine. Many workers have shown that morphine, instead of producing physiological rest to the small bowel, as is commonly believed, produces an increase in tone as well as increased rhythmic contractions.[49,64,65] Some of the beneficial effect of morphine in the early stages probably is due to its lessening the force of respiration, thus diminishing the absorption of bacteria and toxins from the peritoneal cavity.

Morphine, because it stimulates intestinal tone and the rhythmic segmentation movements, is probably of some value in treating the inhibition ileus that occurs in the later stages of peritonitis.

By diminishing reflex activities morphine lessens the tendency to air swallowing, a condition which frequently complicates peritonitis.

In the fully developed peritonitis the depressant effect of morphine upon respiration is not desirable despite the fact that morphine probably diminishes metabolic functions and thus lessens the need of oxygen. When frequent doses of morphine are required the depression of breathing should be counteracted by CO_2-O inhalations.

The experimental work of Reynolds, Arkin and others indicates that substances which have a depressing effect upon oxidation, such as ether and morphine, have a distinct detrimental effect on phagocytosis, as well as other immune reactions.[1,39]

We have all seen cases in which large doses of morphine masked the symptoms and signs of peritonitis, so that removal of the focus of infection and drainage were established too late.

From these observations one can readily see that morphine in moderate doses is indicated, especially in the treatment of the earlier stages of peritonitis, but it seems doubtful that the benefits of deep morphinization outweigh its disadvantages.

RADIOTHERAPY IN EARLY PERITONITIS

Irradiation by means of mild *x*-ray exposures has proved of great benefit in the treatment of many inflammatory lesions, especially when such lesions were in the early, presuppurative stages. It is assumed that irradiation under these conditions, by liberating antibacterial substances from the radiosensitive lymphocytes, raises the defensive powers. If these assumptions are accepted, then mild irradiation should prove of considerable value during the early, formative stages of peritonitis.[67,68]

DUODENAL INTUBATION

The introduction of a Levin duodenal tube through the nose is a valuable therapeutic measure. In the first place it empties the distended stomach and duodenum of swallowed air, a condition which occurs frequently in peritonitis. It drains the excessive secretions that collect in the upper bowel. This is best accomplished by applying slow, gentle suction to the duodenal tube. By lessening intra-abdominal tension, duodenal intubation takes a load off of the diaphragm and improves the splanchnic circulation.

According to Morton there is often a gas bubble at the cardia of the stomach which acts as a trap against ingested fluids. The duodenal tube releases this and lets the patient take fluids freely without having them stick in the lower esophagus. Taking fluids by mouth is a most important prophylactic measure for preventing parotitis, a complication which is serious in peritonitis.

TREATMENT OF INHIBITION ILEUS

Inhibition of intestinal function is early and local in action at first, as witness the loss of tone and motility of the distal ileum in many cases of gangrenous appendicitis. It is possible that this early local inhibition is preceded by a very transitory period of increased intestinal activity due to local irritation of the visceral peritoneum. No effort should be made to overcome the early local inhibition until walling off of the infective process has occurred. For this reason the intravenous administration of hypertonic saline solution to stimulate propulsive motility is to be resorted to only after localization has occurred. The best results from the use of hypertonic salt solution are obtained in those cases where excessive splanchnic inhibition is associated with loss of intestinal tone due to hypochloremia. Pituitrin and eserine are usually ineffective in

the treatment of inhibition ileus because they do not stimulate propulsive motility of the small gut.

TREATMENT OF MECHANICAL INTESTINAL OBSTRUCTION COMPLICATING PERITONITIS

If hypertonic salt solution and duodenal intubation do not relieve the distention, one should consider the possible existence of a mechanical obstruction, which may have to be removed or a decompression-enterostomy may have to be performed. Ordinarily it is deemed inadvisable to break into the area of peritonitis in order to separate an exudate which is producing a mechanical obstruction. A high enterostomy performed in the healthy peritoneal cavity would appear to be the best procedure. Morton, however, reports some excellent results following the removal of obstructions due to plastic peritonitis.[48]

INDICATIONS FOR ENTEROSTOMY

The indications for enterostomy are very limited or of doubtful value in the treatment of diffuse peritonitis, but are well defined in a small percentage of cases of intestinal inhibition associated with matting of loops of the small bowel.[47] Some surgeons perform an ileostomy when an acute appendicitis is associated with obstructive symptoms. The disadvantages of this procedure are the inability to drain distended "paralyzed" loops and, more important probably, the dangers of breaking through the defensive wall.

In many of these cases an enterostomy is not necessary, because the distention and ineffectual motor activity of matted loops is overcome by duodenal intubation and the administration of hypertonic salt solution. Should these fail, then the choice lies between the opening of the appendectomy wound and decompressing a loop of ileum by means of an enterostomy, or doing a higher enterostomy through a high left rectus incision. The latter method is probably the best procedure. One must not forget the deleterious effect of the loss of intestinal juices from a high jejunostomy (despite the administration of Hartmann's solution) and every effort must be made to save these juices by applying a clamp to the enterostomy tube at intervals. In order to avoid as much as possible this high loss of digestive juices it is advisable to do a Witzel-Mayo enterostomy through a loop as far caudad as one can obtain through this incision.

Unless the obstructive symptoms are pronounced and some signs of peristalsis are still apparent, little can be expected from an enterostomy in these cases.

One obstacle to drainage of the small intestine by means of an enterostomy in these cases is kinking of the gut that results from the distention. When the intestine distends, the longitudinal layer of muscle stretches as well as the circular, and the lengthening of the gut results in the production of multiple kinks, which are straightened out with difficulty because of the ineffectual, inhibited intestinal contractions. These multiple kinks can often be overcome by introducing considerable amounts of normal saline solution through the enterostomy and then withdrawing it by means of suction. This should be repeated frequently.

According to some recent experiments of Mensing and Kelley, the stretched longitudinal coat is much slower in recovering its tone after distention is relieved than is the circular muscle.

DRAINAGE IN PERITONITIS

Drainage is of value only in localized cases where the exudate is under tension. Drainage of the peritoneal cavity in cases of generalized peritonitis is generally ineffective and is usually harmful, excepting possibly in children, where there is often a considerable free purulent exudate.

One must not lose sight of the fact that drainage in peritonitis cases is a frequent cause of mechanical intestinal obstruction.

The futility of drainage in general peritonitis is readily seen when it is recalled that the peritoneal cavity cannot be

drained in the ordinary case of general peritonitis and that contamination of the blood stream by bacteria and their toxins had occurred long before drainage was instituted.[8]

ANESTHESIA

Since the sensitivity to trauma of operation and anesthetization is increased in peritonitis and since the induction of anesthesia involves the administration of a toxic substance to a patient who is suffering from bacterial intoxication as well as "toxemia" due to disturbances of chemical equilibrium, anoxemia and the like, the operation that involves the least amount of trauma and the anesthetic that has the fewest toxic effects should be indicated in the treatment of these cases.

The paralyzing effect of spinal anesthesia upon the inhibitory splanchnic nerves produces intense motor activity of the gut and this would contraindicate its use in the earlier formative stages of peritonitis. It is an experimental fact that animals whose splanchnic nerves have been severed succumb quickly of a peritonitis.[24]

Ether is contraindicated because of its deleterious action when a shock syndrome impends, because of its tendency to reverse the intestinal gradient of forces and because it inhibits oxidation processes and thus lessens the immune reactions.

Abdominal block anesthesia, reinforced if necessary by ethylene, appears to be the best anesthetic to use.

SUMMARY

Peritonitis can usually be divided into an *early formative or absorptive stage* during which bacteriemia and bacterial toxemia preponderate, and the *fully developed later stage* in which circulatory disturbances and inhibition ileus preponderate.

The most important factors that enter into the production of symptoms are: (a) bacteriemia and toxemia; (b) dehydration and demineralization; (c) reflex symptoms of nausea, anorexia and general depression; (d) inhibition ileus; (e) circulatory disturbances; (f) anoxemia, and (g) starvation.

The most important local defensive factors against peritoneal infection are phagocytosis, formation of a fibrinous exudate and early localized intestinal inhibition. The general antibacterial activities are interfered with by anhydremia, demineralization, disturbances of the acid-base balance, anoxemia and circulatory disturbances.

The surgical treatment involves the early removal of the focus of infection, with constant consideration of the importance of not disturbing the local defensive mechanisms.

Dehydration and demineralization are treated by means of normal saline, Ringer's and Hartmann's solutions.

The anoxemia is treated by correcting circulatory disturbances and by the early use of oxygen inhalations.

To increase the colloid osmotic pressure of the plasma when a shock syndrome exists, 6 per cent acacia solution with minute doses of pitressin are to be used. (Suprarenal cortex extract may be of some value.)

Fluids are not to be administered by mouth during any stage of peritonitis because they stimulate gut activity. However fluids may be given by mouth during the time that duodenal intubation with suction is applied.

Proctoclysis and enemas are contraindicated in the early cases of peritonitis due to gangrenous appendicitis, when physiologic rest of the cecum is most desirable.

Morphine is needed to control pain. It is doubtful whether deep morphinization has any specific beneficial effect in peritonitis and its deleterious effect upon the respiratory mechanism as well as upon the immune reactions must be borne in mind.

The splanchnic vasomotor paralysis may be treated in the early stages only by means of small doses of ephedrine. Ephedrine also probably lessens "weeping"

from the peritoneum and plasma loss into the intestine, and its inhibitory effect upon gut motility is of advantage during the early stages.

Inhibition ileus and distention are treated by means of duodenal intubation and hypertonic salt solution intravenously. The stimulating effect of hypertonic salt solution upon propulsive intestinal motility contraindicates its use in the early formative stages.

Glucose solutions are especially indicated during the starvation stage.

Fowler's position is of definite value during the early, formative stages. Later the state of the circulation and the patient's comfort should determine the position of the patient.

Mild *x*-ray treatment during the early formative stages of peritonitis is probably indicated because it raises the antibacterial defense mechanisms.

The possibility of a mechanical obstruction occurring in peritonitis, from kinking of the bowel, localized abscess, or from a plastic exudate is to be borne in mind. Enterostomy is indicated only after the simple method of duodenal intubation with suction has been given a trial.

Spinal anesthesia is contraindicated during the early absorptive stages of peritonitis because of its stimulating effect upon peristalsis. Ether is contraindicated during any stage of peritonitis.

The inefficacy of drainage in general peritonitis cases is briefly discussed.

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THE EARLY TREATMENT OF EQUINUS IN CONGENITAL CLUB FOOT*

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THE importance of the equinus component of congenital equinovarus has long been recognized, and the necessity for its adequate correction stressed by many writers on the subject.

That the standard of correction or overcorrection may be at fault is only too often borne out by the unsatisfactory behavior of feet in which treatment was instituted early, and to which the surgeon often had no hesitancy in applying the term "early cure" at the end of the usual manipulative period.

This unsatisfactory behavior may be exhibited to a widely varying degree. There may be a frank tendency to recurrence of the entire deformity necessitating a prolonged battle on the part of the surgeon, the foot oftimes remaining staunch in its refusal to be completely conquered. But more typical are the cases that do not give ready assurance of the eradication of potential recurrence. In the early "follow up" the surgeon does not quite feel sure of the ground gained; especially may this be true at the time the child begins to stand and walk. It appears difficult for him to stand whole soled, and be definitely has trouble in getting the heel down on the floor. The appearance of the feet is not particularly alarming and the varus correction may be well maintained. However inspection reveals the foot to be resistant to dorsal flexion, it cannot be brought up to less than a right angle, and very often considerable force is required to accomplish this. That definite limitation exists can well be confirmed by comparison with the well foot in unilateral cases or with a normal foot in a similarly aged child.

These are the cases in which true overcorrection of the equinus component had not been obtained, and they offer a potential source of supply to that too large group of cases often labelled recurrent or inveterate club foot.

In the treatment of the equinus component various attitudes have been adopted as to the justifiable measures that may be employed for its rectification. In a general way the consensus of opinion is that in the early cases, stretching of the structures producing plantar flexion is sufficient, while the older more resistant ones often need operative interference, notably section of the tendo Achillis.

Division of tendons was practiced early in the seventeenth century by Minnimus and Roonhuysen and in 1816 Delpech is said to have divided the tendo Achilles through a 1 inch incision.[1] Subcutaneous tenotomy of the heel cord was performed by Stromeyer[2] as early as 1833. He reported 2 cases of club foot treated in this manner in 1834.[3] Little[4] introduced the method of Stromeyer in England in 1837 where it became exceedingly popular. Barwell[5] in his treatise on club foot in 1863 decried this popularity, and stated that because of the little danger attached to it "the operation was driven to the worst unbounded exaggeration." Incidentally he goes on to say: "The tendo Achillis requires division by diligent application of ignorant interference to get non-union."

In this country Gross[6] in 1862 advocated tenotomy in the treatment of club foot and said there need be no apprehension about reunion of the ends of the divided tendon. After section he manipulated the foot so as to separate the ends of the divided tendon as far as possible. Adams[7] in his Jacksonian Prize Essay first published in 1866 described extensive experimental work

* Read before the Section of Orthopedic Surgery, N. Y. Academy of Medicine, March, 1933.

on tendon repair both in animals and human beings. He cites examples where after section of the heel cord, no trace

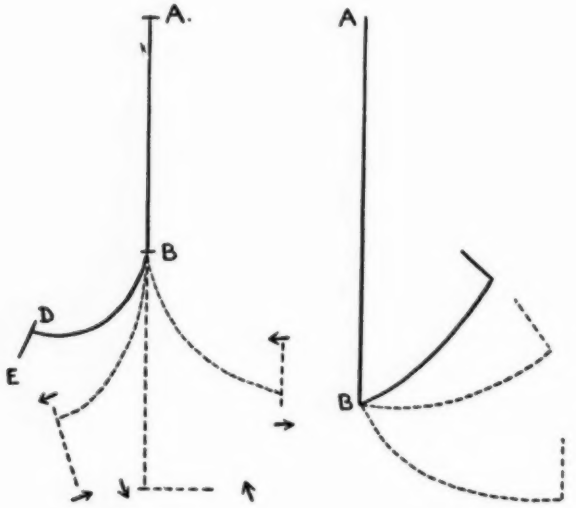

FIG. 1A. FIG. 1B.

FIG. 1. A. Correction of varus component in equinovarus. A.B., long axis of leg. B.D., medial border of foot. D.E., transverse axis of foot or sole line.
B. Correction of equinus component in equinovarus. A.B.D., angle between dorsum of foot and lateral aspect of long axis of leg.

of division could be demonstrated even with the microscope. He called attention to certain cases of club foot with elevation of the os calcis as indicating structural shortening of the gastrocnemius and soleus muscles. In such cases, he stated, the results of mechanical treatment were apt to be uncertain and tenotomy should be resorted to. Bradford[8] in writing on the subject in 1889 believed that correction in infancy and early childhood could be entirely mechanical and the amount of time gained by tenotomy was not great. He stressed, however, the need for full correction of the equinus and its influence on relapse.

Taylor[9] in stating his views in 1892 believed tenotomy more appropriate for the very severe cases. Jones[10] in 1895 commented on the corrective influence of tenotomy on the varus as well as the equinus. The observation has been made that with the persistence of the varus in the heel, the inner border of the tendo Achillis tends to become the shorter one.

Walsham and Hughs[1] in their monograph published in 1895 reported 153 cases of club foot. Non-union had never been observed by them even though the tendon ends were left 1 to 1½ inches apart. Failure to correct the equinus after tenotomy they attributed to structural deformity of the astragalus, namely an abnormal inclination downward of the neck, and the presence of additional bone on the external portion of the neck close to the trochlear surface. This prevented dorsal flexion from occurring in the ankle joint. The specimens upon which they observed these alterations were not young infants.

Eikenbary[11] in 1920 reported 114 cases of club foot and concluded that tenotomy in the manipulative treatment was indicated in mild or moderately severe cases after six months of age.

Elmslie[12] believed better function was to be obtained without tenotomy.

Jones[13] in 1923 stated that division of the tendo Achillis as part of the manipulative treatment should be carried out if the structure was very tight or if the surgeon was afraid of damaging the epiphyses in stretching to correct the equinus.

Ombredanne[14] divides club feet into three classes or periods: complete reductibility, comparative reductibility, complete irreductibility. He believes tenotomy should not be done except as a finishing touch after correction of the varus, and that section of the heel cord offers slight but excellent interference in the second period.

Whitman[15] advocates tenotomy in cases of resistant equinus or in late infancy to save time.

Kite[16] reports successful correction of the equinus by the ingenious wedging of properly applied plaster-of-Paris dressings.

Brockman[17] in his very comprehensive monograph on the subject, concludes the tendo Achillis is best divided by the open method but not until after three years of age.

Massart[18] combines tenotomy with subcutaneous division of the plantar aponeuro-

sis and internal lateral ligament in cases of patients six to eight months of age resistant to manipulation. Roederer[19] likewise be-

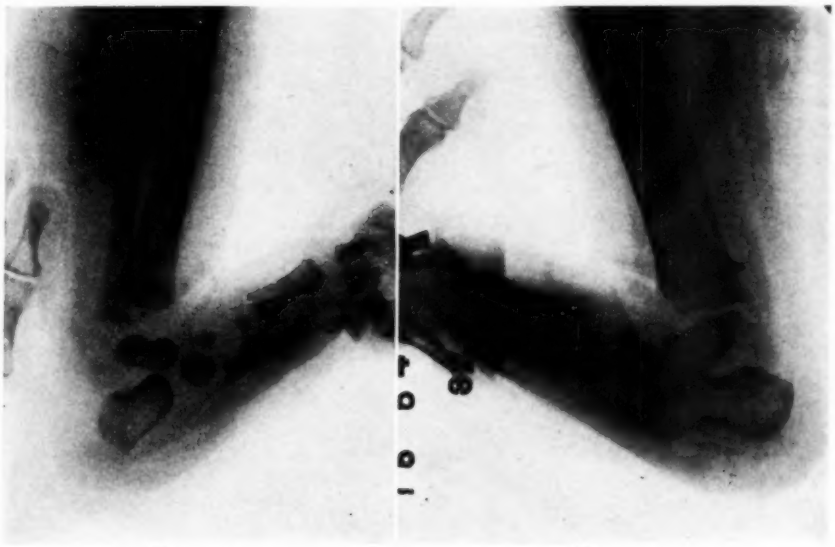

Fig. 2A. Fig. 2B.

Fig. 2. Roentgenograms of two normal feet in position of complete dorsal flexion.

lieves these structures should be sectioned, and that they are the responsible factors in inveterate club foot. Tréves[20] in reviewing the methods of treatment used by various continental surgeons cites the ages advocated by them for tenotomy: Judet, Berger, and Banzet, in the first month; Broca, after five or six weeks; Lamy, three to five months; Froelich after six months and Lucien Michel after ten months if the equinus persists.

Zadeck and Barnett[21] in 1917 reported radiographic studies of clinically overcorrected club feet and called attention to the fact that despite this apparent overcorrection, oftentimes the *x*-ray picture clearly demonstrated the equinus still existent in the os calcis and astragalus. The correction of the equinus had taken place anteriorly to these structures. They found, moreover, that section of the tendo Achillis alone was not sufficient to allow for complete correction of the true equinus, and described a method for subcutaneous division of the posterior ligaments and capsular structures of the ankle joint. However they cautioned against using the procedure soon after manual correction had been attempted. A considerable length of time was allowed to elapse for the midtarsal joints to regain their integrity. The minimum age of the patients constituting their report was one year.[22]

Another factor is to be considered in view of the apparent preference of a varus corrected club foot to become dorsally flexed only in part, on manipulation for correction of the equinus. That factor is the structure of the astragaloscaphoid joint. Brockman[17] in his investigation of the anatomy of the foot has shown the joint to be of a ball and socket type, the head of the astragalus articulating with a variably sized socket made up by the posterior surface of the scaphoid, the sustentaculum and their connecting ligaments. The varying size of the socket, and consequent looseness, or tightness, about this joint depends upon the attitude of the foot, it being diminished in varus and enlarged in valgus.

If such be the case, we have, then, predisposition to anterior dorsal flexion, not only due to resistant structures posterior to the ankle joint, but also to structural properties of the astragaloscaphoid joint.

The purpose of this paper is to acknowledge the merit of the subcutaneous tenotomy and capsulotomy, to show its adoption as a routine detail in the early treatment of congenital club foot, and to demonstrate that in its application, no additional time period, of any moment, is required. In fact in the last seven club foot cases to come under our observation early in infancy, and in which this procedure was carried out, all fixation treatment had been discontinued before the age of six months. Hence in these children there has been and will be a minimum amount of interference with their inclinations to stand and walk at the normal time. This consideration is very often an extremely satisfactory one to the parents. Also, and more important to the surgeon, is the knowledge that true overcorrection of both components of the equinovarus has been obtained. Certainly the appearance, feel, and behavior of such feet warrant some additional feeling of security.

The earlier treatment is started the better, and we agree with Sayre who stated, somewhat idealistically perhaps, that that time was as soon as the baby is washed.

No anesthesia is used for the manipulations in the ordinary early case. Our routine procedure as applied to office or clinic practice may be outlined as follows: Weekly stretchings are carried out for improved position, that is, by manipulations done as gently as possible, the structures are sufficiently stretched to hold the foot in an attitude of inversion and plantar flexion, so that at each sitting a progressive improvement can be safely maintained by the fixation dressing.

All effort is first directed toward the correction of the varus component, and until this is entirely corrected no stretching for the correction of the equinus is attempted. This is for the simple reason

Fig. 3. Club foot in child aged four months. Clinical appearance satisfactory; overcorrection adequate.

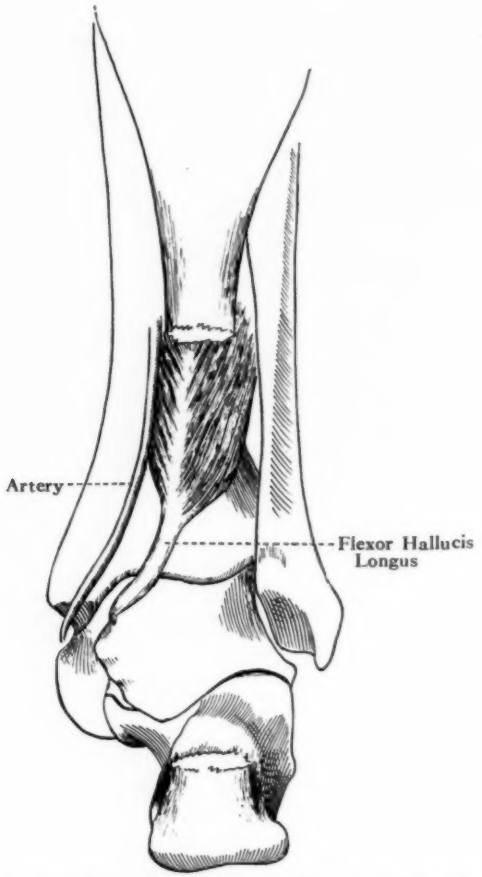

Fig. 4. Section of tendon of flexor longus hallucis in completing capsulotomy.

that the tendo Achillis and posterior ankle structure offer a very usable resistance to work against in stretching for the

Fig. 5. Club foot in same case as Figure 3, one week postoperatively.

correction of the adduction of the foot and inversion of the sole, both elements comprising the varus component. We therefore de-rotate the foot so that the sole line, from one of inversion, progressively and gradually becomes one of normal plane, then one of eversion, and the long axis of the foot progressively and gradually changes from adduction and inward displacement of the forefoot to abduction and outward displacement of the forefoot. The medial border progressively and gradually changes from one of concavity to straightness and then to convexity. The diagram in Figure 1 A demonstrates these stages.

After accomplishing this stage of overcorrection one proceeds progressively and gradually to correct the equinus component by stretching of the tendo Achillis and posterior ankle structures. The upward

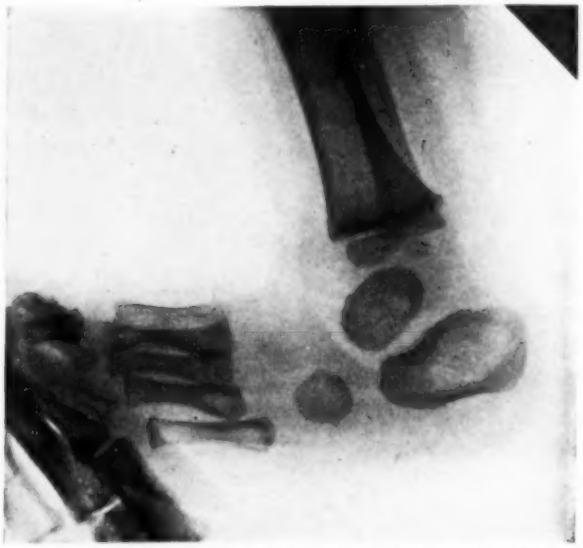

Fig. 6. Roentgenogram of foot in infant aged thirteen months.

pressure is exerted as much as possible against the entire sole. The diagram in Figure 1 B demonstrates this.

By gentle manipulation is meant avoidance of undue injury to the skin or other structures of the foot. Particularly is the skin worthy of the surgeon's greatest interest and respect. Gentle cleansings, followed by alcohol and talcum powder are details not to be neglected. Excoriations, pressure sores or a rash may mean the difference between success and failure in the application of early and adequate treatment.

By progressive improvement is meant definite obvious change in the attitude of the foot at each sitting. Time is valuable. Correction and overcorrection must proceed as rapidly as is consistent with the adaptability of the foot structures to each new position; the circulation and movement response of the toes to tickling constitute obvious indices of safe progress. The number of sittings required varies naturally with the degree of deformity, the resistance and the structure of the foot. In a mild case with a long, narrow,

non-resistant foot, four sessions often are sufficient. In a severe case with a thick, short, tough and resistant foot four times

Fig. 7. Same case after achillotomy.

as many may be required.

The fixation dressing at first is 2 inch plaster-of-Paris bandage applied over a thin layer of cotton flannel bandage, molded evenly and smoothly to the foot. This type of dressing is used until the angle between the dorsum of the foot and the lateral aspect of the long axis of the leg (angle A.B.D. in Fig. 1 B) becomes too acute, with the progress of dorsal flexion. Adhesive plaster may then be effectively substituted, and is so applied that the pull is accentuated against the sole in valgus, and upward in flexion against the leg. No interposition of adhesive takes place in the apex of the angle between the dorsal surfaces of the ankle and foot. Instruction for proper daily stretchings at home may then be instituted, which aid materially in more quickly attaining the end result of overcorrection, an attitude of full calcaneovalgus. The outer border of the foot then lies in close approximation to the lateral aspect of the lower leg. Detailed instructions are

Fig. 8. Foot in child aged twenty months, after manipulative treatment.

always given at the first session to whoever looks after the child at home, in the matter of removal of the fixation dressings. This is to be done at once if any doubt at all arises as to circulatory impairment of the toes.

After full overcorrection has been obtained, a lateral view *x*-ray picture is made with the foot held in complete dorsal flexion. Figure 2 shows a roentgenogram of two normal feet in this position, one in a child aged four and one-half months, and one in a child aged thirteen months. Note the position of the heel bone, it is dorsally flexed along with

the sole line of the foot. Figure 3 represents a club foot in a child aged four months. The clinical appearance was satisfactory, adequate overcorrection. Note here the position of the os calcis. It is at a right angle to the tibia, the dorsal flexion has all taken place anteriorly. The resistance of the tendo Achillis, and the posterior ligamentous and capsular structures hold the posterior part of the foot in relative equinus.

In such a case, the foot as a whole is allowed to come down into a position of equinovalgus and fixed in this attitude for a period of one to two months, depending somewhat on the structural make up, a foot of weak flabby structure possibly requiring more time. In the average foot, two months is ample to acquire sufficient stability at the mid-tarsal joints for the proper leverage at the time of operation. At the end of this period the plaster dressings are removed and a few days allotted for the skin "to breathe" and regain its normal appearance and tone.

Under general anesthesia a subcutaneous achillotomy, with division of the posterior, posterolateral and posteromedial capsular and ligamentous structures is carried out. A single-edged tenotome is used, entry being made medial to the mid-line at the level of the ankle joint. The heel cord is divided from without inward to minimize the possibility of additional skin punctures; then the cutting edge is reversed and entrance made into the ankle joint. Complete division of the capsular and ligamentous tissues is accomplished by working from within outward and from the lateral to the median side. The line of section then approaches the locality of the vessels posterior to the medial malleolus. After the method advocated by Zadek and Barnett, protection is afforded these structures by carrying them toward the skin with the back of the tenotome. Entrance is again made into the joint lateral to the medial malleolus, and the subcutaneous section completed by again working posteriorly.

The tendon of the flexor longus hallucis crosses the posterior aspect of the ankle joint approximately in the mid-line, and

Fig. 9. Same case after subcutaneous achillotomy and capsulotomy.

is necessarily divided in completing the capsulotomy. (Fig. 4.) The section of this tendon, however, is considered of additional benefit as the action of its muscle has been regarded by observers as a pertinent handicap in the maintenance of the varus correction. Certainly it is to be noted that flexion deformities of the great toe are not uncommonly found in club feet.

Injury to the posterior tibial artery has not been experienced by us in this series, but its possibility is to be borne in mind. Several observers have commented that hemorrhage from such an accident can always be controlled by pressure.

If upward pressure on the sole of the foot is maintained during this procedure, audible and palpable giving way of the tissues is apparent. Strong dorsal flexion by considerable force applied as far pos-

teriorly as possible along the sole, completes the operation, and the foot is retained in as much of the corrected attitude as the circulation will permit. Either plaster-of-Paris bandage or adhesive plaster is used; the latter is preferred as more correction is possible. Inspection of the feet within the first twenty-four hours is necessary, and instruction given the parents to cut the straps if signs of circulatory impairment ensue. As a rule these children are allowed to go home the same day.

At the end of one week another forceful stretching is done, but without anesthesia, additional correction being gained at this time. A lateral roentgenogram is then obtained and Figure 5 shows the same foot as Figure 3 at this stage. The equinus of the hind foot has been corrected; the os calcis and astragalus are tilted up anteriorly in line with the dorsal flexion of the entire foot.

The foot is then held in the extreme overcorrected position for one to two months. The proper length of time may be determined by trial periods of recess from the retention dressings, and the tendency displayed by the foot to return to a resistive attitude of previous deformity. The fixation is then discontinued, and daily stretchings performed at home by the parents as indicated.

The application of this procedure to club feet in older children is also to be considered. Figure 6 is the roentgenogram of a foot in an infant aged thirteen months. The parents stated that treatment of the foot was instituted early and had been prolonged. The child had been walking for two and one-half months, but did so awkwardly and the parents were concerned, as an older brother had been similarly afflicted but had not experienced similar awkwardness when beginning to walk. The surgeon treating the younger child proposed operative treatment other than for correction of the equinus. Examination disclosed that the child walked with some in-toeing and inability to bring the heel to the floor. There was moderate varus of the forefoot and marked restriction to dorsal flexion at a few degrees more than a right angle. This is plainly shown in Figure 6. A subcutaneous achillotomy and capsulotomy was performed and the corrected equinus is shown in Figure 7.

Figure 8 demonstrates a foot in a child aged twenty months. This child had bilateral club feet, manipulative treatment was instituted early and at the age of six months he possessed a very satisfactory clinical overcorrection. Observation of the feet at twenty months of age disclosed very satisfactorily maintained correction of the varus, but dorsal flexion completely resisted at 90 degrees. The child had been walking for several months and the mother had noticed a tendency to bear weight on the forefeet; rather than the heels, both in standing and walking. Figure 9 is the roentgenogram after subcutaneous achillotomy and capsulotomy.

CONCLUSIONS

1. A true conception of the existence or non-existence of equinus in a clinically overcorrected club foot can best be demonstrated by the *x*-ray.

2. A large percentage of cases of congenital talipes equinovarus, treated early, need division of the tendo Achillis and posterior capsular and ligamentous structures for the correction of the equinus.

3. This procedure can be done adequately and safely by the subcutaneous method.

4. Structural deformity of the tarsus in line with the equinovarus equation can only exert its minimal influence in feet in which early complete overcorrection has been carried out.

A subsequent report of the feet referred to in this paper is anticipated in the future.

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* Continued from p. 460.

OSTEITIS DEFORMANS AFFECTING THE BONES OF THE FACE*

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OSTEITIS deformans is a chronic inflammatory disease, which generally affects the long bones, the bones of the pelvis, the spinal column and the cranium, but which seldom affects the bones of the face. We are reporting 5 cases of this disease with involvement of the bones of the face, which are not of the leontiasis ossea type. The disease most often afflicts patients who are in later life, and is characterized by destruction of bone, and formation of bone in different planes, and with enlargement of the part.

Knaggs stated as his belief that the reason the facial bones are commonly thought to escape this disease is probably because they are slightly involved; thus they evade notice. He cited 2 cases, one a case of Turner's, in which the superior maxilla was so enlarged that the unaffected alveolar margin of the lower jaw fitted completely and easily within it. The other was a case of Gordon Watson's, in which both jaws were considerably enlarged as part of a generalized condition.

Cayley, in 1878, described a case of hyperostosis of the lower jaw, parietal bone, left clavicle and tibiae, which suggested the possibility of Paget's disease with facial involvement. Beck, in 1907, described a typical case of Paget's disease of the face. Higbee and Ellis, in 1911, described a case of generalized osteitis deformans in which there were changes in the bones of the face. The deformity involved the right malar bone, the right nasal bone, and the right superior and inferior maxillae. Abbe, in 1918, stated that in 14 of his cases of this disease seen in recent years, the jaw was involved in 4. Hamburger and Nachlas, in 1926, described a case which they considered to be osteitis deformans, and which they felt should be distinguished from leontiasis ossea.

ETIOLOGY

The etiology of osteitis deformans is unknown. Many hypotheses have been advanced to explain the cause of this condition, but most of them have been disproved. Locke mentioned nine possible causes. Lozer described cases in which osteoporosis, osteomalacia, or rachitis formerly had been present, and on which osteitis deformans later became superimposed. Most writers have stated as their belief that disturbances of the endocrine glands are generally responsible for the condition. Recently, the hypothesis of faulty calcium metabolism resulting from parathyroid deficiency has received much attention in this field. Knaggs presented an hypothesis based on ideas of toxicity, and his view has been supported by the pathologic observations of Adami and the dietary treatment carried out by Tubby. Rabinowitch stated as his belief that osteitis deformans belongs to the so-called metabolic diseases rather than to the infectious disorders.

SYMPTOMS AND SIGNS

Osteitis deformans is a disease of middle age or advanced life. The youngest authentic patient on record was aged twenty-one years; the case was reported by Maizord and Baurges. The disease is characterized by its insidious onset and slow progress. The patient first feels below par for a long time before definite symptoms appear. The first and most constant symptom is pain, which generally starts in the lower

* Read before the Midwestern Section of the American Laryngological, Rhinological and Otological Society, Iowa City, Ia., Jan. 10, 1933.

extremities. However, about 20 per cent of the patients, according to Bortz, have no subjective symptoms.

In the later stages of the disease, neuromuscular and cardiovascular symptoms appear. The patient may complain of

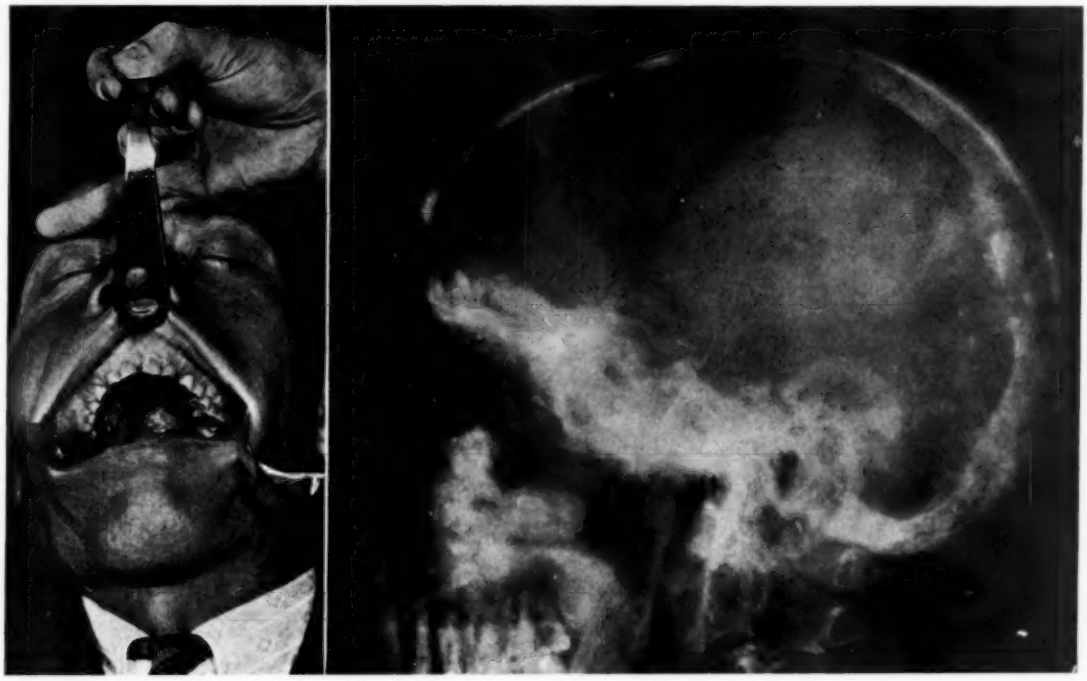

Fig. 1A. Fig. 1B.

Fig. 1. A, Osteitis deformans. Enlargement of upper jaw and palate may be noted. B, characteristic roentgenologic findings of upper jaw and skull.

When the bones of the face are involved, the disease may involve the entire superior maxilla, as in Case I, or the involvement may be unilateral as in Case II, or confined to one region, forming a small bony protuberance, as in Case III. As the condition advances, there appears definite increase in the size of the bones, particularly those of the skull, the clavicles, the long bones and the bones of the pelvis. The bones of the legs become bowed anteroposteriorly. The spinal column is curved anteriorly, so that the abdomen is reduced in size, and the costal margin approaches the pelvic brim. Accompanying the changes in bone there is generally marked weakness which gives the patient an appearance of premature senility. Carman and Carrick described the characteristic appearance and posture: "The head, which is increased in size, is carried forward and shaped like an inverted triangle."

headache, vertigo, fainting, pain, paresthesia, paralysis or incontinence of urine and feces. Mental symptoms develop in some, in the form of hysteria, depression or dementia.

ROENTGENOLOGIC OBSERVATIONS

Roentgen rays constitute the most reliable agent for diagnosing osteitis deformans. Generally many bones are involved. The order of frequency of involvement is tibia, femur, skull, pelvis, spinal column, clavicle, ribs, radius and ulna, whereas the bones of the hands, feet, or face are rarely affected.[7] The roentgenogram is characteristic (Fig. 1A and B). In the early stages only the proximal portions of the shaft of a given bone may be involved.[16] As the disease progresses the narrow space gives a mottled appearance, and the whole bone becomes much larger, due to the very marked subperiosteal thickening and

formation of new bone. The new bone is irregular in distribution, causing the outline to be indistinct, and giving a fuzzy, cloudy, or smoky appearance. Cystic spaces may occur, but they are rarely more than 0.5 cm. in diameter.

Fig. 2. Hyperparathyroidism; characteristic picture of skull.

The distinction between osteitis deformans and hyperparathyroidism involving bone must be considered (Fig. 2). Camp stated:

> The fundamental change in hyperparathyroidism is revealed in the majority of cases as uniform, miliary, granular osteoporosis. This peculiar form of mottled atrophy is distinct from the ordinary type seen in osteoporosis associated with acute and chronic disease of bone and neurotrophic conditions. In my experience there is no other generalized skeletal lesion which exactly simulates the miliary mottling and granular appearance of the bone in hyperparathyroidism, and such an appearance seems to be characteristic of this condition.

Camp also stated that formation of cysts in this disease is seen in the mandible in some cases, and gives rise to the predominating symptoms. Leontiasis ossea (Fig. 3), osteitis fibrosa cystica, certain osteofibromas and giant-cell tumors (Fig. 4) give roentgenographic pictures which might, under certain conditions, need to be distinguished from osteitis deformans. Osteomas, too, if they occur bilaterally, might present a problem in differential diagnosis (Figs. 5 and 6).

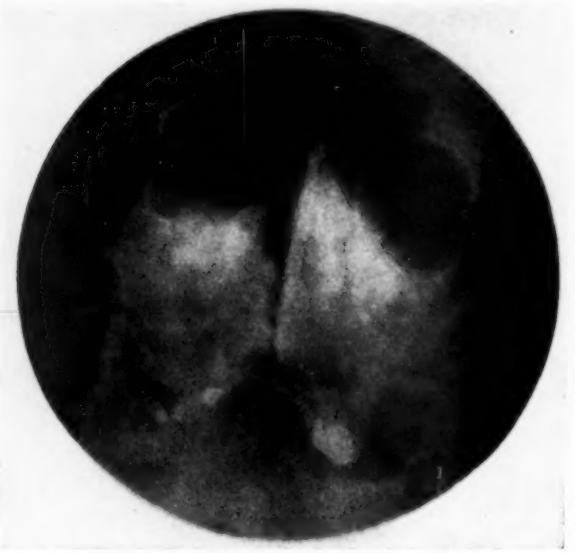

Fig. 3. Leontiasis ossei; almost complete obstruction of both nostrils.

PATHOLOGY

The general process starts with engorgement of the vessels and destruction of the original bone, which is replaced by soft, cancellous bone. The new formation is greatly in excess of the original bone, so that the bones become much thicker and even longer, but more irregular in outline. During the early stages the softness of the replaced osseous tissue causes deformity of the long bones, which follow the normal lines of curvature. As the disease progresses the newly formed osseous tissue becomes dense, hard, and sclerosed, so that it is even more resistant than normal bone.

Bloodgood and Knaggs expressed doubt whether cystic spaces are ever present in true osteitis deformans, and said that cases in which true cysts can be demonstrated are probably cases of osteitis fibrosa cystica. Locke said: "Rarely small spaces are seen in the shaft, seldom more than 1 or 2 cm. in diameter, filled with reddish gelatinous material. These are the bone cysts which occur with such a variety of bone diseases. These are always

small in size, and may be single or multiple." Camp stated as his belief that there are two types of osteitis deformans: in

Fig. 4. Giant-cell tumor of upper jaw.

one, there is predominating fibrous change, whereas in the other, as in Case 1, there are predominating cystic changes.

Histologically, the same general process may be seen. The rarefaction and destruction of the bone start beneath the periosteum and progress centrally. The destructive process is accompanied by formation of new bone from the newly formed connective tissue which has replaced the original bone. Knaggs pointed out the similarity of the histopathologic picture in osteitis deformans and osteitis fibrosa, but mentioned several points which serve to distinguish osteitis deformans microscopically. In osteitis deformans there is a regular and even distribution of the new trabeculae which are separated by comparatively narrow tracts of osteogenic tissue. The trabeculae reveal internal curvilinear markings as a result of the new bone being laid down on the original bony trabeculae. There is evidence of fat cells in most of the sections, and infiltration of small lymphocytes is present.

DIAGNOSIS

Several conditions should be particularly considered in the differential diagnosis of osteitis deformans, especially when it occurs in the bones of the face. These conditions are leontiasis ossea, osteitis fibrosa, hyperparathyroidism, multiple

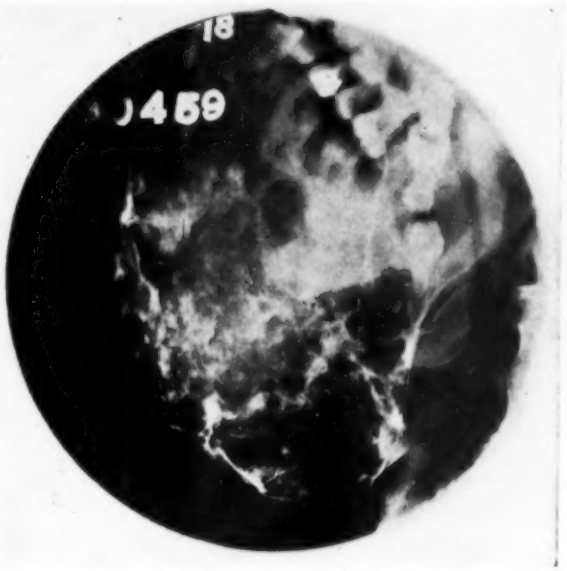

Fig. 5. Fibro-osteoma of lower jaw.

myeloma, giant-cell tumor and fibro-osteoma.

Knaggs stated that osteitis fibrosa and osteitis deformans are simply different expressions of the same disease, determined by dissimilar resisting powers of the victims. The generalized form of osteitis fibrosa is the most difficult condition to distinguish from osteitis deformans. However, osteitis fibrosa begins much earlier in life (in the first or second decade), whereas osteitis deformans generally begins in the fourth to fifth decade and progresses slowly but steadily. Fractures occur quite commonly in osteitis fibrosa, but rarely in osteitis deformans. Formation of cysts is more characteristic of osteitis fibrosa than of osteitis deformans. The pathologic process is fundamentally similar in the two conditions, especially in the earlier stages, as mentioned.

Leontiasis ossea is a term which has been used to designate almost any type of overgrowth of the facial bones. Prince, in 1902, said that a distinction between hyperostosis cranii and osteitis deformans was to be found in the hypertrophy of the malar

bone in leontiasis ossea, but that this was not always found in this disease and conversely the bones of the upper part of the face are not always unaffected in Paget's disease. He expressed the belief that there was no sure ground for distinguishing hyperostosis cranii from osteitis deformans.

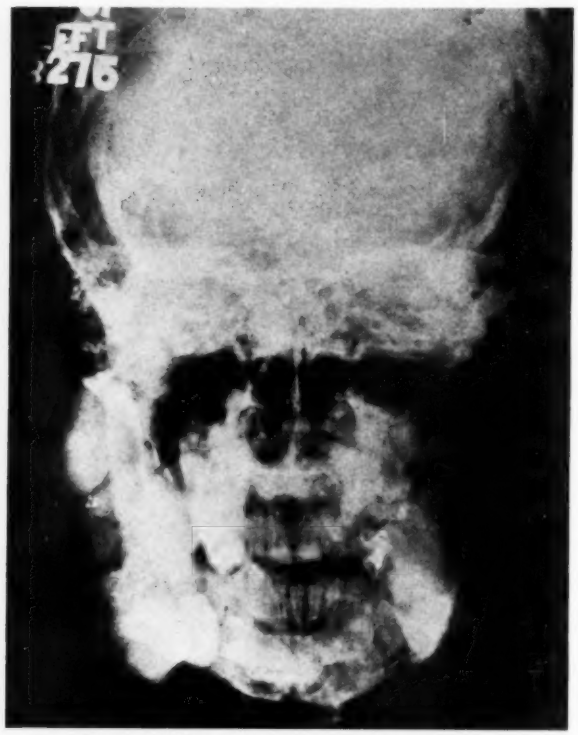

Fig. 6. Bilateral osteomas of lower jaw.

Many writers have described typical cases of osteitis deformans under the heading of leontiasis ossea. However, the typical picture of leontiasis ossea as described by Knaggs is certainly separate and distinct from osteitis deformans with facial involvement. First, it starts early in life; second, other bones of the body are not involved, and, third, the roentgenologic findings differ.

TREATMENT AND PROGNOSIS

Patients with osteitis deformans generally live many years, and often die from other causes. There is no satisfactory treatment that will alter the course of the disease. Symptomatic treatment for relief of pain is about all that can be done other than improving the patient's appearance by removing a sufficient amount of the enlarged bone to make the face as nearly normal as possible. If the nose is obstructed by the enlargement, the obstructing bone should be removed, and a rubber tube inserted in each nostril in order that an airway may develop for breathing in the normal way. In Turner's case, improvement followed removal of teeth and clearing of infection of the jaw. Kanavel advised surgery in leontiasis ossea. Abbe advised and described a surgical procedure for removal of bony overgrowth in Paget's disease of the face.

REPORT OF CASES

Case 1. A married painter, aged thirty-seven years, registered at The Mayo Clinic on January 9, 1922. In 1913, when the patient was twenty-eight years of age, a small cavity in the second upper right molar tooth had been filled. After the filling he had experienced severe sharp pains which had radiated to the right temporal region. The pain had been accompanied by swelling of the gum margins on the right. In November, 1921, he had been struck on the right side of the face by a piece of wood. Two weeks later a painful swelling had appeared in the region of the upper right second molar tooth. Although the pain gradually had disappeared, the swelling had persisted and had been accompanied by a purulent discharge.

Examination, January 12, revealed a hard tumor of the right upper jaw, involving the palate and alveolar process, with bulging of the right cheek. There was a sinus above the right upper teeth from which pus was discharged. The upper teeth on the right side were loose.

The tumor of the jaw was explored February 10, 1922, and was found to be hard over most of the surface. Above the right upper molar teeth there was a soft region which when explored was found to be a cyst extending to the floor of the nose and into the cavity of the antrum. Soft, bone-like tissue composed the greatest part of the tumor, the inside of which was removed. Malignancy was not found.

The patient returned for observation at intervals of six months, but little change was

noted until March, 1930, when a sinus formed in the right side of the upper jaw, above the alveolar process. A considerable amount of thick pus drained from this sinus. It was enlarged with a curet and was found to extend deeply into the body of the jaw. The tumor had increased markedly in size since the patient's first visit in 1922.

In 1922 a roentgenogram of the upper jaw revealed marked thickening, which was thought to be caused by a chondroma. However, by 1930 the condition had advanced so that it was readily recognized by means of roentgen rays as evidence of extensive Paget's disease, involving all of the bones of the skull, with rather marked osteomatous formation over the right mandible. Further roentgenograms revealed that there was extensive Paget's disease of the entire spinal column, with predominating cystic changes and flattening of many vertebrae. The pelvis and upper part of the femurs also gave evidence of this condition, with predominating cystic changes. All of the long bones were involved.

Case II. A married woman, aged fifty years, registered October 9, 1929. Nine years previous to examination a hard lump had appeared in the left side of the upper jaw in the region of the molar teeth, and had gradually enlarged. All of the molar teeth on that side had been removed because of the lesion. In October, 1928, an attempt had been made elsewhere to remove the tumor by means of escharotics, which had resulted in an opening being made into the left antrum. Soon after this complete surgical excision had been attempted. Secondary closure two weeks later had been unsuccessful. For the next seven months the patient had been treated with radium.

When the patient was examined at The Mayo Clinic it was found that the left maxilla had been removed as far forward as the left central incisor tooth, and an opening 2 cm. in diameter extended through the cheek over the antral region. Also, through the floor of the orbit, there was a smaller opening which was about 0.5 cm. in diameter. Roentgenograms of the sinuses and head revealed that Paget's disease involved the bones of the head and face, with clouding of all of the sinuses. Further roentgenograms revealed that Paget's disease involved the bones of the pelvis and both femurs. The patient was not examined further nor was treatment given.

Case III. A single woman, aged sixty-five years, registered October 5, 1925. In March, 1924, a routine examination of the patient's teeth, made elsewhere, had revealed no abnormality. In February, 1925, her dentist had discovered a bony prominence on the upper jaw.

When the patient was examined at the Clinic in October, 1925, the bridge of her nose was found to be thickened, and there was an enlargement about 4 cm. in diameter in the median line of the upper maxilla. Roentgenograms of the skull at that time suggested early Paget's disease.

When the patient returned, in 1927, the lesion on the superior maxilla was definitely enlarged. In 1930 there was further increase in size, with bony prominences in the frontal and left parietal regions. Roentgenograms of the head revealed the presence of Paget's disease involving the skull and bones of the face, with involvement of the sphenoid bone and diminution in the size of the sella turcica. Other roentgenograms gave negative results. The patient was not examined further nor was treatment given.

Case IV. A married man, aged sixty-two years, entered the Clinic January 6, 1921. His only complaint was of pain of two years' duration in the lumbar region, right hip, and knee.

Roentgen examination revealed the presence of Paget's disease of the upper part of the femur, spinal column and bones of the pelvis. Further roentgenograms taken of the head revealed Paget's disease of the skull, malar bone and inferior maxilla.

Case V. A married man, aged fifty-two years, entered the Clinic May 16, 1923. He complained of pain, of one year's duration, in the left side of the face. The pain came in paroxysms, was not affected by heat or cold, and was not relieved by taking of sedatives. He also had difficulty in swallowing. He noticed that he had had to buy larger hats from year to year, and that "lumps" had been present in his head for many years.

Examination revealed weakness and atrophy of the muscles in the left temporal region and around the left shoulder. On the left side of the nasopharynx, just above the eustachian tube, there was bulging without ulceration. A roentgenogram revealed the presence of Paget's disease of the skull with marked in-

crease in density along the floor of the sphenoid bone.

CONCLUSIONS

We have presented 5 cases of osteitis deformans in which the bones of the face were involved. Although involvement of the facial bones is rare in this condition, other cases have been reported but generally under the title of leontiasis ossea. In most of the cases of this type there is associated involvement of other bones of the body. The course of the disease is prolonged, and little can be done for the patient to stop its progress, but when the bones of the face are involved, operative removal for cosmetic purposes is well worth while.

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A METHOD OF TREATMENT OF

UPWARD DISLOCATION OF THE ACROMIAL END OF THE CLAVICLE*

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UPWARD dislocation of the acromial end of the clavicle occurs fairly frequently. In the case of a complete dislocation the acromial process of the scapula is forced downward and the coracoid process upwards, thereby rupturing the coracoclavicular ligament as well as the acromioclavicular ligaments, and dislocating the acromial end of the clavicle upward. When the traumatizing force is not great there is an incomplete dislocation.

FIG. 1. Pad of cotton and saddler's felt placed over dislocated end of clavicle and held in place by adhesive plaster.

FIG. 2. Elastic bandage placed about shoulders under tension in figure of eight and in such a manner as to cover and compress pad and underlying dislocated end of clavicle. Axillae should be padded with cotton.

Deformity is evident following this injury. There is a prominence visible at the acromioclavicular joint and a drooping of the shoulder. Movements of the shoulder are limited and painful. The dislocation of the clavicle can be palpated.

An *x*-ray picture of the acromioclavicular joint may fail to show a dislocation when the exposure of the film is made with the patient reclining and tending to reduce the dislocation. In some patients there is a considerable space normally present between the interarticular ends which may seem to exceed the average separation of 1 cm. It is evident that the most valuable diagnostic film is secured by making pictures of the injured and of the normal shoulder for comparison.

Usually the dislocation may be readily reduced by manipulation. However, after the bones are approximated they are not easily held firmly in place until healing of the joint has occurred. Inadequate retention of the reduction is likely to result in disability due to the insecurity of the shoulder region when in motion.

To reduce the dislocation it is necessary to lift the arm and scapula upward above their normal level, backward, and make sufficient downward pressure on the outer end of the clavicle to hold it in place. Many procedures have been reported as giving satisfactory fixation. A common

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method of attempting to immobilize the shoulder in this position is by the use of a sling or adhesive tape that passes around

Fig. 3. Arm on injured side placed in sling to displace weight of extremity from shoulder and to lift scapula upwards.

underneath the elbow of the injured side and crosses over the outer half of the clavicle. In this method the weight of the arm supplies the downward traction on the clavicle, but in this manipulation the entire shoulder is forced forward and downward, thus making for recurrence of the dislocation. Furthermore, the downward tension on the clavicle at the site of dislocation is relaxed by movements of the arm and forearm. More effective tension on the clavicle can be made by joining two parts of an adhesive sling together by a strong piece of rubber. Shaar[1] applies the tension in the same manner by the application of a canvas splint and rubber tubing traction. These methods of traction however offer only partial control and as in the first method described tend to force the shoulder forward and downward. Dillehunt[2] secures retention of the shoulder in position by incasing the arm of the affected side in a plaster cast and attaching this cast to a plaster jacket that encircles the trunk.

The rational position for fixation of the shoulder after reduction of an upward dislocation of the outer end of the clavicle is backward and upward. A simple and comfortable method of securing this position is accomplished by the use of an elastic bandage placed about the shoulders in a figure of eight. Before the elastic bandage is applied a pad of cotton and saddle felt of 1 or 2 inch thickness and about 2 inches in diameter is placed over the outer end of the dislocated clavicle and held in place by adhesive plaster. (Fig. 1.) The axillary folds and spaces are padded with cotton for comfort and to absorb perspiration. The elastic bandage is applied fairly tensely about the shoulders in the manner of a figure of eight and so as to make pressure on the pad overlying the outer end of the dislocated clavicle. It is desirable to use a bandage containing rubber. The elastic bandage pulls the scapulae and hence the acromion processes backward and upward and at the same time effectively depresses the pad and the dislocated end of the clavicle. (Fig. 2.) The bandage is prevented from slipping by the insertion of safety pins at points where the bandage crosses itself. The tension of the elastic bandage is made more effective by placing the forearm on the injured side in a fairly tight sling. The sling displaces the weight of the extremity from the shoulder to about the neck and helps to elevate the shoulder above its normal level. (Fig. 3.) An axillary pad is used to prevent inward displacement of the shoulder.

Fixation by this means should be made for four to six weeks. The elastic bandage can be removed occasionally to allow tightening when replaced and to permit cleansing and powdering of the parts. After the use of the elastic bandage is discontinued the arm should be carried in a sling until complete healing has occurred. This method of fixation has been used satisfactorily on patients and it is believed that it will materially reduce the number of patients on whom it is necessary to perform an open operation for cure of the dislocation by arthrodesis, fixation by suture or by other means.

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AVERTIN ANESTHESIA IN BONE AND JOINT SURGERY

FROM THE SURGEON'S VIEWPOINT*

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AN extensive literature is now available regarding the use and effect of avertin, tribromethyl alcohol, anesthesia in various fields of surgery. The advantages and disadvantages of this anesthetic have been widely discussed. It is the purpose of this paper to comment upon the use of avertin in bone and joint surgery. The following report is based upon avertin anesthesia in 250 consecutive operations.

Surgical procedures in the bone and joint field are mainly confined to the extremities and to the spine. These operations are often time consuming, particularly since there so commonly follows application of plaster jackets, casts, or placing the affected extremity in suspension and traction, during all of which time it is essential that the patient be anesthetized. Deep anesthesia to the degree which is required for work in the abdomen is rarely necessary, because in abdominal surgery by traction on the mesentery and irritation of the diaphragm the operator excites the strong deep reflexes, which are not disturbed in the usual bone and joint procedures. However, muscle relaxation is essential and must be further maintained during application of plaster, or placing the limb in suspension and traction. If Balkan frame apparatus is to be utilized, the patient, of course, has to be returned to bed. In this case, the anesthetist accompanying the patient must continue anesthesia and that in turn requires taking a cumbersome gas machine along to the ward. If inhalation ether is employed the ward atmosphere becomes permeated with the fumes, which are extremely annoying to the other patients. Spinal anesthesia is very satisfactory in operations on the soft parts of the lower extremities, but is not equally good when bone work is done. The patient is conscious of the shock of the mallet and this reacts upon him in an unpleasant manner.

It is the writer's opinion that avertin employed as a basal anesthetic best meets the requirements for anesthesia in bone and joint surgery. So decided are its advantages that avertin is now the standard anesthetic for all operations on the Bone and Joint Service at the Clinic. The reasons for this choice are:

1. *Ease of induction.* It can be administered by any competent nurse under the anesthetist's direction. The patient experiences no discomfort and there is complete absence of the usual preoperative apprehension. Consequently this anesthetic is an ideal one for children.

2. *Postoperative recovery is smooth; the patient is comfortable.* This smooth recovery has consistently been the case, particularly where avertin anesthesia alone was necessary, or in patients requiring a relatively small amount of gas. (Avertin anesthesia can be employed as the sole anesthetic in minor surgical procedures, especially in complicated painful dressings.) Repeatedly on awakening patients have desired to know when the operation will be performed.

3. *There have been no deleterious effects from the anesthetic.* Three patients complained of slight rectal tenesmus, but there was no blood or mucus in the stools, and the symptoms disappeared within two or three hours following the return of consciousness.

An initial drop in blood pressure has been reported by the anesthetist in many instances, immediately following the intro-

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duction of the anesthetic. This finding has been discussed at length in the literature, but in none of our cases has it proved to be a persistent drop. On their return to bed the patients have uniformly been in good condition.

There has been no clinical evidence of impairment of liver function. The efficiency of the liver function was tested by determining the urobilinogen in the urine, the normal value being one part of urobilinogen in 20 parts of urine. In this connection one patient is of particular interest. He was a somewhat unstable boy of seventeen, markedly sensitive to pain and fearfully apprehensive, suffering from an extensive acute suppurative osteomyelitis of the upper end of the femur, which involved the hip joint. Because of this fear and inability to withstand discomfort it was necessary to administer an anesthetic even for some of the minor dressings. In a space covering two years' time this youth had twenty different operative procedures, for each of which he was given avertin. The urobilinogen has remained normal.

Another patient was a girl, two years of age when first seen, suffering from an acute suppurative osteomyelitis of the lower end of the femur, with an additional infection of the knee joint. This child was submitted to various procedures requiring an anesthetic, a total of sixteen times, yet the urobilinogen is normal.

4. *Excellent control of patient throughout the operation.* At any time during the operative procedure should the stimulus resulting therefrom unduly arouse the patient, it is a simple matter to administer or to increase the amount of nitrous oxide or ethylene as the case may be, and secure the necessary degree of relaxation. When this is done the patient does not resist the introduction of the accessory anesthesia, so that from the surgeon's standpoint there is no interference with the operation.

In any operation of major character, avertin is always employed simply as a basal anesthetic. The accessory gas anesthesia is begun at the start of the operation. Should greater relaxation be necessary, it is easily obtained by increasing the gas concentration. At no time has it been necessary in any of the procedures in which this anesthetic was used to employ ether as an additional anesthetic. The accessory anesthetics were always either nitrous oxide or ethylene.

DOSAGE

As administered by the Anesthesia Department under Dr. Lincoln F. Sise, the details are as follows:

At the present time avertin fluid is employed, 1 c.c. of which is equivalent to 1 gm. of the drug. After the dosage is calculated (see below) the anesthetic is administered by rectum in 2½ per cent solution, with distilled water colored with congo red as the diluent.

Preliminary medication is now simply that of a hypodermic of morphia, the dosage naturally depending on the age and size of the patient, varying, for adults, from ¼ to ⅙ grain. In some instances atropine, $\frac{1}{200}$ to $\frac{1}{150}$ grain, is also given, particularly if the weather is very warm and it is felt that the patient would perspire excessively.

In young children of good healthy physique the average dose is 100 mg. per kilogram body weight, while in somewhat older children who are also well developed and quite husky the dose may be raised to as high as 120 mg. per kilogram body weight.

In young adults of vigorous constitution, or similar types of patients, the average dose is 100 mg. per kilogram. In the average adult patient, such as one in the middle age group, of ordinary health and vigor, the dose is about 80 mg. per kilogram body weight.

In the very old and quite feeble, the dose is as a rule around 60 mg. per kilogram, supplemented to a greater degree with nitrous oxide or ethylene. In a few instances a dose as low as 40 mg. has been utilized, but with very little effect.

The anesthetic is administered twenty minutes to one-half hour before the operation begins, preferably in the patient's room. The average duration of the full anesthesia is one to one and a half hours, followed by relatively deep sleep for six or more hours, during which the patient can be aroused if desired. Throughout this period of deep sleep it is extremely important that a free airway be maintained so that there can be no possibility of even a slight degree of anoxemia.

During the postoperative period the amount of medication so commonly necessary is markedly cut down. Frequently patients go through the first eighteen to twenty-four hours with no postoperative sedative whatever. I am still further impressed by the patient's ability within or immediately after the first twenty-four hours to resume his usual diet, and to exhibit relatively little or no effect from his surgical experience.

COMMENT

Avertin anesthesia is especially well adapted to bone and joint surgery as contrasted with its use in general surgical practice, because avertin gives a somewhat prolonged, not very deep, anesthesia with good muscular relaxation; a duration and degree of relaxation which exactly meets the surgeon's requirements. Furthermore, avertin alone suffices for the postoperative application of plaster casts or the fitting of suspension and traction apparatus.

Finally, it is of decided importance to appreciate the fact that as contrasted with avertin or avertin and gas, an equally long gas or gas and ether anesthetic causes much more pronounced postoperative nausea, vomiting and general upset.

A CHEMICAL ATTACK ON CANCER*

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EACH year about 100,000 people die from cancer in this country. The cancer death rate is not decreasing; if anything it seems to increase slightly. Cancer, therefore, is one of the most important of our public health problems.

The human body is composed of a marvelously well-organized agglomeration of millions of different types of microscopic units, called cells. The view held most prevalently is that cancer is a disease of some of these cells which causes their unrestricted growth and multiplication and their destructive action on the surrounding normal cells. Cancer usually starts as a *localized* disease, but sooner or later some of the diseased cells are carried by the blood or lymph to other parts of the body and the disease becomes generalized.

The real *cause of cancer* is still unknown. There is good reason to believe that cancer is not an infection caused by a specific germ. We do know, however, that long-continued irritation of the tissues of the body may produce cancer. Typical cancer has been produced in animals and man by coal tar and certain pure chemicals, by *x*-rays and radium, and by certain parasites.

The most desirable method of controlling cancer is of course its *prevention*. This, unfortunately, can be done only in a limited way. For instance, it is perfectly feasible to prevent so-called "occupational cancer" in people who are continuously exposed for years to *x*-rays, radium, coal tar or shale oil.

Early recognition of the disease, when it is still localized, offers good hope for a *cure* by removal of all cancer cells by surgery or their destruction by *x*-rays or radium. Unfortunately the methods for early recognition are far from satisfactory and in a large percentage of patients the disease has progressed too far for a cure by present methods of local treatment. In my opinion the outstanding needs for a better control of cancer are, first, new methods for early recognition, and second, effective methods for curing the disease in a more advanced stage.

We have attacked the problem from the curative viewpoint. We are fully aware of the tremendous difficulties which must be overcome, but the outlook for some success is by no means hopeless, if sufficient thought, energy and time are applied to the problem. Chemistry has contributed so effectively to the advance of medical knowledge that we can assume it will also help in the solution of the cancer problem. So far no chemical treatment of cancer has been shown to be of practical use. To achieve this it is better, in our opinion, to study first some of the fundamental aspects of the problem. As in engineering so also in medicine the great practical forward steps have been made possible by fundamental discoveries.

We have not concerned ourselves with the scientific explanation as to how and why a normal cell is converted into a cancer cell. This most important question, dealing with the real cause of cancer, will have to wait until we know a great deal more not only of cancer cells but also of normal cells. But we feel that chemistry will be of help in throwing light on the *nature of the cancer process and that such knowledge will in time suggest a chemical method for controlling the disease.* The history of medicine clearly shows that it is not always necessary to know the cause of a disease in order to discover an effective treatment. Sometimes it is sufficient to

* Presented before the Science Forum of the Electrical Society of New York, Nov. 16, 1932.

know enough about the disease process. The treatment of pernicious anemia with liver extract is a striking illustration of this fact. The modern, rational surgical treatment of cancer was largely based on laboratory work on animals, which showed that cancer begins as a *local* disease. The *x*-ray and radium treatments were more or less accidental discoveries.

Chemistry of Growth and Division of Cells. I have already pointed out that the unrestricted growth and multiplication of cancer cells is one of the outstanding characteristics of this disease. A man who has spent his life in the study of cancer has said that "the problem of the nature of cancer is probably identical with that of growth itself." It is therefore of the utmost importance *to discover chemicals which in small doses will inhibit the multiplication of malignant cells without causing serious injury to the normal body cells.* This is our ultimate aim. Now we know very little about the kind of chemicals which control the growth and division of normal or cancer cells. The problem is extremely difficult because the living cell is the most complex organization of matter known.

To insure accurate chemical control of the experimental conditions we have started work first on a normal cell—Amoeba. We have found that glutathione, a substance containing sulphur and occurring in most animal cells, when added to the cells in very small amounts, stimulates their division. As we have found this substance also in cancerous tissue it is reasonable to suppose that it also plays a part in the growth of that tissue. On the other hand, copper salts, which also regularly occur in cells, were found in infinitesimal amounts to inhibit cell division. Similar doses of iron, manganese, cobalt and zinc were without appreciable effects. We also found that small amounts of lead salts inhibit cell division. This is of interest in connection with the claims made for the recent lead treatment of cancer, which, while it may have some slight value, is far too dangerous to the patient to be of general usefulness.

Work is also under way with cultures of normal and cancer cells in glass vessels. We have devised methods for controlling the carbonic acid and oxygen tension of cultures and the nutritional requirements of these cultures and their response to chemicals are under investigation.

Cleavage and Synthesis of Cell Protein. Cells naturally cannot grow and multiply indefinitely without the building up of protein, for protein is part of the structure of all cells. Protein is made up of the largest and most complex molecules known and determines to a considerable degree the specific properties of different cells. We have put ourselves the question as to what chemical conditions favor either the building up or the breaking down of cell protein. It has been known for a long time that some organic substances (the enzymes or ferments) can break down protein in animal cells, but until very recently it was only a reasonable assumption, unsupported by experimental proof, that these enzymes may also build up protein and thus permit cell and tissue growth. A year ago we found that the breaking-down of the protein of certain normal and cancerous tissues in the test tube is favored by reducing the oxygen pressure. This suggested that a high oxygen pressure under otherwise identical conditions might cause the building up of protein from its split products. This we found to be the case. Leaving all other conditions unchanged, it was possible to break down protein in an atmosphere of nitrogen, to build it up again by admitting oxygen and to reverse the process once more in nitrogen.

This fundamental observation may possibly have a far-reaching influence on our understanding of the cancer process, as oxygen is indispensable to man and the higher animals. Under normal conditions its supply to the organs, where it is utilized, is carefully controlled through the circulating blood. Hence growth is well

regulated and cells do not die under these conditions. In some portions of the cancerous growth the blood, and therefore the oxygen supply, may be adequate for the building up of protein and therefore for the growth and multiplication of cells. In other portions of the cancerous tissue, however, the blood and oxygen supply may easily become inadequate and in this way cause a breaking-down of cell protein, resulting in an inhibition of growth. Such considerations may partly explain the well-established fact that growth, multiplication and death of cancer cells occur *simultaneously* in the same malignant tumor.

Acidity of Cancerous Tissue as a Cause of Its Destructive Action on Invaded Normal Tissue. I have called attention to the fact that cancerous tissue has a destructive action on the surrounding invaded normal tissue. The question is: can this destructive action be explained on a chemical basis? Our work is based on the following facts. The starch (and sugar) in the food is the main source of energy of the human body. This starch is changed into sugar during digestion. The sugar is burnt in the body to carbonic acid and water. Incomplete combustion of sugar yields lactic acid. Cancerous tissue produces excessive amounts of lactic acid. The question is: *does lactic acid accumulate in malignant tumors and invaded normal tissue?* If so, this lactic acid may cause a local toxic effect and may lead to the destruction of cells; because present information indicates that under physiological conditions the acidity of cells and tissues is carefully adjusted to a narrow range compatible with life. We were able to devise a suitable method for studying this problem. An electrode, consisting of a fine glass capillary, sealed off at one end and filled with a suitable solution, is inserted into the cancerous or normal tissue of living animals. The electrode is then connected with a suitable measuring equipment (electrometer). In this way we found that the malignant tissue has the highest acidity, the invaded tissue less, and the normal tissue least. Quite recently we have been able to show that the injection of certain sugars into animals afflicted with transplanted or spontaneous tumors is rapidly followed by a very pronounced increase in the acidity and lactic acid content of the cancerous tissues. These results, therefore, indicate that acid accumulates sufficiently in the tumor and surrounding tissue to shift the acidity to a higher value. The conditions for a local toxic action of this increased acid appear to be fulfilled.

To sum up it will be evident that our efforts are directed towards a better understanding of the chemical conditions which cause on the one hand the continued growth and multiplication of normal and cancer cells; on the other hand their inhibition of growth and multiplication and their death. Our observations indicate that glutathione and copper have a powerful action on cell growth and cell multiplication of a normal type of cell, that the oxygen supply to the cells is one of the factors which controls the building up and breaking down of cell protein, and that acid apparently accumulates in cancerous tissue and surrounding invaded normal tissue so as to cause a local toxic effect on both cancer and normal cells. We hope that this kind of knowledge concerning the fundamental nature of the cancer process will suggest in time chemical methods for controlling cancerous growth.

Note: The non-technical wording of this paper was necessitated by the fact that it was presented to a lay audience.

THE EARLY DIAGNOSIS OF CARCINOMA OF THE LARGE INTESTINE

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THE cure of colonic and rectal carcinoma depends largely on early diagnosis, followed by radical extirpation of diseased and adjacent tissues. Our therapeutic limitations in the treatment of far-advanced lesions make early recognition of malignant lesions in such situations of the utmost importance. For this reason 400 cases of proved carcinoma of the colon and rectum, selected from recent records of The Mayo Clinic, were reviewed for the purpose of more accurate evaluation of early symptoms. An equal number of cases (100) from each of four situations was selected: (1) cecum and ascending colon; (2) transverse colon; (3) descending colon and sigmoid, and (4) rectosigmoid and rectum. For the sake of clearness, the most common initial symptoms of carcinoma in these four segments of the large bowel will be considered individually. The typical syndromes pathognomonic of advanced growths will not be considered.

In accord with present views regarding "cancer-bearing tissue," it is of interest that 2.7 per cent of the 400 patients had had some type of malignant neoplasm removed prior to discovery of the intestinal lesion. Approximately two-thirds of the patients were men, and the average age of all patients was fifty-three and two-tenths years.

CECUM AND ASCENDING COLON

The misdirected surgical treatment which these patients received during the period of symptoms ascribable to the neoplasm of the colon seems worthy of mention. Fifteen per cent of this group of 100 patients had been subjected to appendectomy. One patient had had an operation on the biliary tract, and on four an exploratory laparotomy had been performed within two years prior to their visit to the Clinic. Diagnostic errors of this magnitude are now seldom justifiable. Prior to recent developments in roentgenology, the diagnosis of malignant growths, as well as of chronic inflammatory granulomatous lesions of the cecum and ascending colon, was notoriously difficult.

The presence of a mass in the lower right abdominal quadrant, or unexplained anemia, were common prerequisites to detection of a cecal tumor. Although diagnostic criteria and laboratory aids have greatly facilitated earlier recognition of lesions in this region, yet all too frequently the patient is seen when changes are far advanced and irreparable before an accurate diagnosis has been made. This may be explained in part by the fact that the right half of the colon is a relatively "silent" area, giving rise to symptoms only after considerable alteration in structure or function has occurred. Lesions in this portion of the colon remain more difficult to detect early than those more distally situated.

In this series of cases, pain was by far the most common initial complaint of 59 patients; 86 patients complained of pain at some time during the course of their illness. The pain varied considerably both in type and situation, but most commonly was a more or less constant dull ache in the right lower abdominal quadrant. Attacks of pain simulating subacute appendicitis, beginning with generalized abdominal distress and later localizing over the region of the cecum, also occurred. Occasionally there was slight tenderness in this region, but actual rigidity, associated leucocytosis, or fever was not noted. The pain

was never severe enough to necessitate morphine for relief. Rarely was it diffuse in the lower part of the abdomen, and rarely in the epigastrium. Although at times the distress caused by carcinoma of the cecum may be referred along the right costal margin around to the back, it should not be mistaken for the pain of cholecystic disease, as it is less severe and has no associated symptoms suggestive of biliary disorder. The rare occurrence of attacks suggesting intestinal obstruction is explained by the large lumen and liquid content of the proximal portion of the colon.

The second most common initial complaint which was noted by 13 patients was that of tumor in the lower right abdominal quadrant. Although a mass of this type did not occur as a primary indication of trouble in many cases, it constituted one of the first three complaints of 29 patients, and it was found on examination of 62 patients. The disagreeable corollary, when masses are grossly palpable prior to surgical intervention, is the realization of delayed diagnosis. Granted that a definite proportion of tumors in the cecum and ascending colon may be entirely symptomless for several months following their inception, undoubtedly many of them are suggestive of their true nature, which might obviate the loss of valuable time if the significance of this evidence were properly appreciated. A tumor of this type is palpated as a rounded, usually rather fixed, slightly tender, deep-lying mass in the lower right quadrant.

Third in order of prominence as an initial complaint in cases of malignancy of the right side of the colon was indigestion. Although it was one of the first three symptoms of 38 patients, it was noted as the primary disturbance of only 10. The symptoms ranged from anorexia, gas pains, and flatulence to actual nausea and vomiting. Although at first this indigestion may be intermittent, it often becomes constant as time progresses. Among approximately half of the patients, when first observed at the Clinic, some type of digestive disorder existed. Not uncommonly the loss of appetite, which frequently persists for some time following extirpation of the growth, suffices to account for considerable loss of weight. Seldom does this indigestion mimic a definite clinical syndrome characteristic of pathologic change elsewhere in the gastrointestinal tract. It is far more likely to be indefinite, with the result that it is all too readily explained on a functional basis.

Eighteen patients had various less common initial symptoms, among which were change in the habit of bowel movement, passage of blood or mucus from the rectum, pallor and loss of strength or weight. Although these symptoms are uncommon as an initial warning of a malignant growth in the right half of the colon, such a growth should always be considered in the absence of a satisfactory explanation for the symptoms. Sometimes malignancy is discovered only during the course of general physical examination. The duration of symptoms prior to the establishment of an accurate diagnosis was found to be less than one year in 57 per cent of cases, one to two years in 28 per cent, and two to ten years in the remainder.

Examination of the patient with carcinoma of the cecum or ascending colon was usually of little aid in making an early diagnosis, except that 42 patients had moderate tenderness in the lower right quadrant of the abdomen which might not be associated with a palpable mass. The fact that 91 of the patients had lost weight, ranging from 10 to 40 pounds, may have aided in the recognition of a hitherto undetected growth. A palpable mass was found in 62 cases, marked anemia was often apparent, but these symptoms contributed little to the establishment of an early diagnosis.

The early recognition of carcinoma in this region is rendered difficult, not so much by inadequate clinical methods, as by absence of commonly understood lo-

calizing symptoms. The examining physician's lack of suspicion accounts for many of the errors. There are certain definite symptoms and complaints which should always demand careful investigation of the colon before they are dismissed as being inconsequential. Among these are atypical appendiceal pain which runs a rather chronic course or recurs frequently for a considerable period; failure to obtain relief of symptoms after appendectomy; vague abdominal pains of any nature which tend to localize in the lower right quadrant; local tenderness which is atypical for appendicitis; unexplained dyspepsia; unexplained anemia, and loss of weight or strength without apparent cause. The occurrence of these symptoms in a patient of mature years may be the first warning of serious disease, and if suspicion is aroused it may result in the diagnosis of carcinoma of the cecum. Anemia, loss of weight and a palpable tumor should be considered late diagnostic criteria. The most common erroneous diagnoses are primary anemia, carcinoma of the stomach, appendicitis, "intestinal flu" and cholecystic disease.

TRANSVERSE COLON

Carcinoma of the colon distal to the hepatic flexure is less prone to masquerade under indefinite signs, or to imitate other maladies. Despite this, attention is not called to many neoplasms of the caudal two-thirds of the colon, or they remain undiscovered until far advanced.

Pain is by far the most frequent initial symptom caused by a growth in the transverse colon; it occurred in 49 cases. In contrast with the pain of lesions of the right side of the colon, distress from a growth in the transverse colon is more likely to be intermittent and like a colic. Attacks of pain often occur after meals and may be relieved by passage of a stool or gas. The relation to food may be so marked as to simulate the syndrome of peptic ulcer, and occasionally such an erroneous diagnosis is made. The most typical attacks, however, suggest mild evanescent obstruction. More severe attacks may be associated with vomiting. The pain is usually generalized throughout the abdomen, perhaps centering around the umbilicus, but it may be high, low, on either side or even referred along the costal margin or to the rectum. Morphine occasionally is necessary for relief. This is uncommon, although the pain of these lesions is usually more severe than of cecal lesions.

Definite obstructive attacks constituted the next most common primary symptom. This occurred in 17 cases, and was one of the first three symptoms in 31 per cent of the cases. These attacks were characterized by moderate to severe abdominal pains relieved only by stool, and were unassociated with the excessive vomiting and disturbed chemical constituents of the blood which accompany obstruction higher in the gastrointestinal tract. The attacks varied in duration and intensity, but gave pathognomonic evidence of colonic obstruction and should have aroused suspicion of their etiology. Acute obstruction requiring emergency surgical decompression rarely occurred.

Gradually increasing constipation was the third most common initial symptom; it occurred in 13 cases, and developed later in 32 cases.

Blood in the stools, loss of strength or weight, indigestion, palpable tumor or diarrhea, was the first warning symptom in the remainder of the cases. Practically all patients had lost weight by the time surgical treatment was instituted. The indigestion was characterized for the most part by belching, bloating, borborygmus, anorexia, nausea or vomiting. Forty-four per cent of the patients complained of gas in one form at some time during the course of the disease. A palpable tumor was detected by 20 per cent of the patients before they sought medical aid, and was discovered in 49 per cent of the cases on physical examination at the clinic. Eleven of the patients complained of

diarrhea. Contrary to what might be expected, the duration of symptoms before surgical treatment was only slightly less than for lesions of the cecum and ascending colon. Physical examination of these patients was generally of little value in making an early diagnosis. In approximately half of the cases, a palpable tumor was found, and abdominal tenderness was elicited in 20 per cent. In some cases there was abdominal distention. As a rule, loss of weight is not so marked as among patients with malignancy of the cecum.

DESCENDING COLON AND SIGMOID

Malignant growths of the descending colon and sigmoid are more prone to present symptoms widely recognized as characteristic of carcinoma of the colon than lesions situated proximal to the splenic flexure. Malignancy in this region, in contrast to that elsewhere in the colon, affords more definite localizing signs. The contents of this portion of the large bowel are usually formed, and in the presence of an encircling lesion readily cause obstruction or increasing constipation. It is this type of syndrome that is generally associated with neoplasm of the large bowel, with the result that carcinoma in this region does not usually present the preliminary diagnostic difficulties often encountered with growths elsewhere in the colon.

The most common initial symptom was pain, which occurred in 40 of the cases. A total of 75 per cent of patients with growths in this region experienced pain at one time or another. This is likely to be more severe than if lesions are on the right side, and is often present more or less constantly as a dull ache with periodic exacerbations of acute cramps. In the majority of cases it is situated in the lower or left side of the abdomen, but may be generalized and not definitely localized. Rarely is it situated in the upper part of the abdomen or even on the right side.

In this series of cases, increasing constipation or evidence of obstruction was the second most common initial complaint occurring in 37 cases. Increasing constipation occurred first in 24 per cent and developed sooner or later in 54 per cent. Obstructive attacks constituted the initial warning in 13 per cent of the cases; these usually yielded to medical treatment, but occasionally required surgical decompression. Although there is commonly an antecedent history of constipation, acute complete obstruction may suddenly develop without any previous warning. In fact, such an occurrence is most common in this portion of the large bowel, although lesions of the transverse colon may at times exhibit a similar acute onset. Diarrhea occurred in 12 cases. A change in bowel habit occurred in 70 per cent of the cases; occasionally the character of the stool changed.

Blood in the stool was the third most common initial symptom; this occurred in 18 cases. It was observed early or late, associated or unassociated with mucus, in 50 per cent of the cases. Unfortunately, as is so often the case with lesions of the rectum or rectosigmoid, the warning symptom was often neglected, and was considered to be caused by hemorrhoids. Less commonly, digestive disturbances such as anorexia, dyspepsia or gas were noted first, and at least one of three patients had some disturbance of this nature during the course of the illness. Other primary complaints were unusual, but at times loss of strength or weight, or irritability of the urinary bladder gave warning signs. The duration of symptoms in these cases was similar to that for lesions of the transverse colon.

Physical examination may be of little aid in making an early diagnosis. Abdominal tenderness, found in at least 30 per cent of the cases, may or may not be associated with the presence of a palpable tumor. Distention is observed usually as an accompaniment of obstructive attacks. Approximately 88 per cent of the patients

lose weight, which is less prone to be extreme than when the lesion is situated in the right half of the colon.

RECTOSIGMOID AND RECTUM

The diagnosis of carcinoma of the rectosigmoid and rectum is far easier than of malignancy in any other portion of the large bowel. Notwithstanding this fact, errors in diagnosis are almost as common in this segment of bowel as they are in that segment proximal to the sigmoid. The reason for this may largely be attributed to inadequate examination and willingness on the part of many physicians to assume that hemorrhoids, fissures or fistulae are accountable for most rectal disorders. Even if such mild ailments are more prevalent than carcinoma, it still remains indisputable that every patient who passes bright blood from the rectum should have the benefit of proctoscopic examination, and if the results of this are negative, a roentgenogram of the colon should be made. To appreciate this precept, it need only be realized that 10 per cent of all patients suffering from carcinoma of the rectum and rectosigmoid that are seen at the Clinic have had some operative procedure, performed elsewhere, on the rectum for a minor disorder when symptoms of malignant disease were active.

Change in the bowel habit, which has usually been long and well established, is the most common original symptom; it occurred in 47 cases of carcinoma. This change may be characterized either by constipation or by diarrhea. Often one may start and later give way to the other. The diarrhea, which is usually bloody may be associated with considerable tenesmus and small passages of stools which may be comprised chiefly of mucus, débris and blood without much fecal matter. Colitis is often diagnosed and medical treatment is instituted. More commonly, when constipation occurs, the character of the stool may be altered, either being flattened (ribbon stool) or in small firm lumps. Any alteration in a long and well-established habit of evacuation should at once demand thorough investigation of the entire large bowel, if the cause is not found in the rectum or rectosigmoid. In this series, 89 per cent of the patients experienced either constipation or diarrhea, or both, alternately before they sought operation.

Rectal bleeding was the first symptom in 39 cases, and occurred either with or without discharge of mucus in 96 per cent of the cases. This varied in degree and occurrence from spotting, discovered at the conclusion of defecation or between stools, to bright blood on the stool and even severe intermittent rectal hemorrhages. More than half of the patients noting this type of discharge attributed the trouble to hemorrhoids. The most pathognomonic syndrome of carcinoma of the rectum was increasing constipation associated with rectal bleeding.

The third most frequent early symptom was pain; this occurred in 10 cases. It was seldom severe, and changed from the rectum and pelvis to the sacral region and the lower part of the abdomen. Occasionally dyschesia was noted. At one time or another 63 per cent of the patients complained of pain. This was usually mild and was often described as a soreness or an ache. Digestive disturbances rarely gave premonitory warnings of serious disease.

The only really valuable procedure in the physical examination of these patients consists of digital examination of the rectum. This alone should suffice to make a fairly certain diagnosis in practically 100 per cent of all cases of malignancy of the rectum. Loss of weight or strength may be entirely absent or slight.

Actual visualization of the existent change in the rectum or rectosigmoid through a proctoscope and the histologic appearance of the specimen removed will make the diagnosis certain. Eighteen per cent of these patients had coexisting well-developed hemorrhoids, which further emphasizes the fact that if the diagnosis of bleeding due to the hemorrhoids is made, it should be made only by exclusion.

COMMENT

The symptoms of carcinoma of the colon and rectum seem worthy of emphasis, not because they enable one to make a prompt diagnosis without further investigation, but primarily because they direct attention toward the probable site of disease and afford the necessary clues which demand a thorough study of the large bowel. The diagnosis of malignant growths in the colon by purely clinical means is at best uncertain.

The remarkable development of roentgenology during recent years has been largely responsible for the greater accuracy with which these lesions are recognized. The use of the barium enema in the hands of skilled roentgenologists now permits a correct diagnosis of colonic neoplasms situated proximal to the sigmoid flexure in more than 95 per cent of cases. With so accurate a diagnostic procedure, detection of early lesions as well as exclusion of suspected lesions has been materially facilitated.

Lesions situated in the sigmoid and below it may be readily detected either by digital or proctoscopic examination. A specimen removed for biopsy to determine the grade of malignancy is of great value in determining operability, and is easily obtained when the lesion is visualized through the proctoscope.

SUMMARY

The earliest ascertainable clues and symptoms of carcinoma of the colon and rectum have been considered, and factors aiding in an early and positive diagnosis have been reiterated.

The most common initial symptoms of malignant growths of the right side of the colon are pain, palpable tumor and indefinite dyspepsia. Lesions in the transverse colon manifest themselves first by pain, obstructive attacks and developing or increasing constipation. Carcinoma of the descending colon and sigmoid is manifested first by increasing constipation or obstruction, pain, rectal bleeding and diarrhea. Carcinoma of the rectosigmoid and rectum is suggested by a change in bowel habit, either constipation or diarrhea, and rectal bleeding. The diagnosis of malignant lesions of the colon is rendered certain by roentgenographic examination. The presence of lesions in the sigmoid or below it may be proved by proctoscopic and digital examination of the rectum.

VALIDITY OF PRESENT CRITERIA FOR THE DIAGNOSIS OF CAROTID BODY TUMOR

(WITH ESPECIAL REFERENCE TO BRANCHIOGENETIC CYSTS)*

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PERHAPS one of the most uncommon as well as one of the most difficult tumors to diagnose is that of carotid body tumor. Although a review of the literature reveals 196 cases, many more patients have been operated on. Unfortunately, not all of these have been put on record, for we have knowledge of 5 cases in our relatively small circle of friends, none of which has found its way into the literature. In Bevan and McCarthy's excellent addition to this subject, the statement is made that few men have had the opportunity to see a case, and that no man has had more than three. Recently, however, Rankin and Wellbrock reported 12 cases from the Mayo Clinic, by far the largest series yet reported. The correct preoperative diagnosis has been made approximately twenty times.

Since Marchand, in 1891, reported the first case it has been considered a diagnostic accomplishment to make a correct preoperative diagnosis. When presented with a swelling on the lateral side of the neck, the possibility of a carotid body tumor is ordinarily rarely considered.

For the past twenty-five years evidence and certain criteria have gradually accumulated leading to the diagnosis of carotid body tumor. In 1906 the inimitable Keen, who published one of the first papers in America on this subject, laid down the structure leading to such a diagnosis. He states:

> The diagnosis is based on positive and negative facts. The positive facts are: 1. There is a tumor arising in the region of the bifurcation of the carotid, that is to say, about at a level with the upper border of the thyroid cartilage. It lies at first under the border of the sternocleidomastoid, but when it has reached any size it is only partially covered by this muscle. 2. The tumor is only moderately movable horizontally but not vertically. 3. It is usually ovoidal in shape. 4. As a rule it is rather smooth and not lobulated. 5. It is a single tumor and not made up of a coalescence of several tumors, as in tuberculous glands. 6. There is transmitted but non-expansile pulsation. 7. There may be a bruit and thrill, but this is rare and not marked. 8. It may cause bulging of the wall of the pharynx. 9. The pupil is occasionally contracted. 10. It will usually grow slowly at first, and, possibly shortly before the surgeon sees it more or less rapidly. 11. It may have existed for a number of years. 12. The consistence is neither very hard nor very soft, but elastic.
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> The negative facts: 1. There is no pain. 2. As a rule it is not tender. 3. There is no disturbance, as a rule, of the pneumogastric, either as to its cardiac or laryngeal function. 4. There is no change in the pupil, except occasionally. 5. There is no disturbance of the sympathetic nerve. 6. So far as the patient is concerned, there are usually no subjective symptoms, but the aid of the surgeon is sought on account of a slowly or rapidly growing deformity.

Bevan writes:

> With the evidence we now have and which has been accumulating in the last twenty years, the clinical diagnosis will be made more frequently in the future from three points: the location of the tumor at the crotch of the carotid; the sense of pulsation in the tumor; and the fact that the tumor is movable from side to side but is not movable from above downward; and the added fact that the tumor has existed for many months or years before the patient seeks surgical relief. . . . In all cases a visible tumor mass is present usually in the upper anterior cervical triangle with the pos-

* From the Tumor Clinic, Department of Surgery, Northwestern University Medical School.

terior margin under the sternocleidomastoid muscle. The tumor mass is of variable size, some being as small as a hazelnut, others as large as a goose egg. Usually they are of hen's egg size when the patient first consults a physician. The mass practically always pulsates but the pulsation is not that full expansion and contraction of an aneurism but rather a sort of transmitted pulsation. In most cases pressure on the mass will considerably diminish its size, since this forces the blood out of the highly vascular mass. There may be a thrill and a bruit. The skin of the neck is unchanged and it is freely movable over the mass. The mass is movable laterally but not up and down, and is almost always attached to the surrounding structures. The surface is usually smooth but in several cases it has been nodular. Practically never do patients with carotid tumors complain of pain, although there may be occasionally tingling sensations in the neck. The mass is not tender to palpation.

Rankin who reports the largest collection of such tumors lists practically the same findings. Regarding the pulsation he adds, "Almost always the pulsation is present, but lacks the expansile quality of an aneurism, and although there is no pathognomonic symptom, the pulsation is typical."

Our interest in this interesting tumor was precipitated by a young man who presented himself to the Tumor Clinic at the Northwestern University Medical School.

The patient was twenty-four years old, and complained of a painless, non-tender mass in the neck of four months' duration. Soon after its discovery (it was first noted by a relative) the mass grew rapidly for a month until it reached the size of a hen's egg, after which it remained practically stationary. The mass was on the right side, in the region of the bifurcation of the carotid, that is about on a level with the upper border of the thyroid cartilage, partly covered by the sternocleidomastoid muscle. The posterior portion of the tumor was underneath the muscle. The skin over the mass was unchanged and freely movable. The tumor was oval, smooth, firm but elastic, with no evidence of fluctuation. It was freely movable from side to side but immovable up and down. There was a distinct transmitted but non-expansile pulsation, but no thrill or bruit was present. There were no other palpable glands in the neck. The right pupil was smaller than its mate, and pressure on the tumor produced a prompt dilatation of the pupil. There was, in addition, the phenomenon of hippus, particularly present when a strong light was thrown into his eyes at which time there was a rapid rhythmic contraction and dilatation of the pupil. The general physical examination was otherwise negative as were the laboratory findings.

Here then was a case which had fulfilled all the requirements necessary to make a diagnosis of tumor of the carotid body, with perhaps the period of its duration as the only dissenting factor. The tumor mass was ideally situated; it was firm but elastic; it was freely movable horizontally but not movable vertically; it had a distinct transmitted pulsation; it was painless and not tender; the skin over the mass was normal and unattached; it had grown rapidly and in addition showed signs of irritation of the cervical sympathetic.

A diagnosis of carotid body tumor was made and operation was advised. In suggesting this we were not unmindful of the magnitude of such a procedure. We were cognizant of the frequent necessity of ligating one or all of the carotid vessels, with its concomitant, tremendous morbidity and mortality. We were not unfamiliar with the frequent occurrence of hemiplegia, aphasia, aphonia, and the many other permanent complications which resulted in many of the non-fatal cases. We were aware, also, of the fact that a majority of these tumors were benign, and we knew that one case (Bevan's) was considered cured by *x*-ray therapy. We were aware, also, that a number of these tumors, especially in young people, had been successfully removed without complications and without the necessity of sacrificing any of the important vessels.

These facts were carefully weighed and explained to the patient. He was given to understand that we would not place his life in jeopardy; that if at operation we found it neces-

sary to ligate any of the carotid vessels, except the external carotid, we would not proceed with the operation. He consented and on November 26, 1932, under local anesthesia, an incision along the anterior border of the sternocleidomastoid was made. The muscle was found considerably thinned and beneath it could be seen, much plainer now, the pulsating mass. After this muscle was drawn to one side, and after a little blunt dissection we came upon the tumor mass which was the size of an hen's egg. It was grayish brown, elastic, but not as firm as when palpated through the skin. The capsule was rather thick and the mass was bound to the surrounding structures, especially at its lateral and posterior surfaces. By the use of blunt and sharp dissection the mass was dissected free from its bindings. It was found, however, to be intimately attached to the internal jugular vein, from which it had to be carefully shaved off. The posterior portion of the tumor lay in the bifurcation of the carotid but not intimately attached to these vessels. The tumor was completely shelled out and recognized as a branchiogenetic cyst. The young man made an uneventful recovery. The cyst was filled with a yellowish brown, soft, thick, liquid material and showed large quantities of cholesterol in the form of crystals. Sections from the wall showed a thick stratified squamous epithelium lining the inner surface as well as the papillary projections. Underneath the squamous epithelium there was a thick layer of lymphoid tissue with follicles and germinal centers, followed, towards the periphery, by a loose connective tissue diffusely infiltrated with plasma cells, lymphocytes and leucocytes, mainly eosinophilic.

As we reviewed this case in retrospect, the justification in arriving at a diagnosis of carotid body tumor with the available evidence now on hand was questioned. A review of the literature will soon reveal the astounding facts that branchiogenetic cysts will in every way mimic tumors of the carotid body. The great majority of these cysts are situated in the region where carotid tumors occur, lying in the region of the bifurcation of the carotid and partially covered by the sternocleidomastoid muscle.

While it is true that perhaps the bulk of cysts are soft or even fluctuate, it is not uncommon for these tumors to be firm and elastic. This is especially true in the so-called atheromatous type, which are densely filled and which give the elastic sensation. Carotid body tumors, likewise, may vary in consistency, some portions being hard in contrast to the general softness of the tumor. In Keen's case the tumor was so soft that he regarded it as a lipoma, and explored it as such.

The statement is usually made that branchial cysts are rather freely movable unless previous inflammation has fixed them to the surrounding structures. Most of them, however, give the sensation of being attached rather loosely to the deep structures. As the great bulk of these cysts extend downwards and become attached to the sheath of the internal jugular vein, vertical mobility will be difficult or impossible. At times such cysts become intimately attached to the sheath of the carotid, and again, a cyst may straddle the vessel at its bifurcation and become attached to all three arteries. It is in the uncommon type, those that lie underneath the sternocleidomastoid muscle but above the deep vessels, that free mobility may be elicited. This latter type is rare as compared to the type which passes down and lies on the great vessels. Senn, in 1844, asserted that only lateral motion was possible, on account of their peculiar attachment to the deep structures of the neck.

Although uncommon, transmitted pulsation is known to occur in branchial cysts. In Hyndman and Light's collection of 108 cases, in Carp and Stout's 20 cases, and in Bailey's 63 cases, as well as many other collected reports of branchial cysts, no mention is made of this finding. Bailey further adds that there is no record of a case of branchial cyst which was pulsatile from transmitted impulse of the carotids. This latter statement of Bailey's probably refers to his group of cases, for in 1884 Senn in an excellent paper on "Branchial Cysts" says:

Branchial cysts of the second and third clefts are always observed in the sheath of the large cervical vessels, usually in the carotid triangle above the omohyoid muscle. If the tumor is only of moderate size, the pulsation of the carotid artery can be felt on its inner margin. If it is larger, it overlaps the artery, when the pulsations of the vessel are communicated to the tumor.

He mentions also several other cases of pulsatile branchial cysts appearing in the literature up to that time. Little or no mention is made of this phenomenon in recent literature, although it is well worth remembering. This finding, plus the lateral mobility and absent vertical mobility, in our case, was a great factor in leading us to an incorrect diagnosis.

Secondary symptoms, as a result of pressure, have been occasionally noted in carotid body tumors. Likewise have such findings been present in branchial cysts. Dysphagia has been reported in both types of tumors. Paralysis of the recurrent laryngeal nerve has been reported in cases of carotid body tumor. Thompson reports a case of branchial cyst associated with huskiness of the voice and on laryngoscopic examination one of the vocal cords was found immobile. Soon dyspnea and aphonia became prominent requiring immediate intervention. With the removal of the cyst the voice returned to normal as did the previously fixed vocal cord.

It has been noted several times that pressure on a carotid body tumor will considerably diminish its size, since this forces the blood out of the highly vascular tumor. This finding has been noted in large branchial cysts with deep ramifications. By emptying into the retropharyngeal spaces, such cysts are known to decrease markedly in size; therefore while exercising this pressure, the pharynx should be watched before pronouncing the "sign of emptying" positive (Bailey).

With our present knowledge can we with any degree of assurance arrive at a correct preoperative diagnosis of tumor of the carotid body? Branchial cysts and carotid body tumors may have, as we have shown, identical histories and findings. These two conditions may so closely simulate one another as to be indistinguishable. Bailey has adequately shown that the presence of cholesterol crystals on microscopic examination of the aspirated contents is conclusive evidence in making a diagnosis of branchial cyst. Aspiration can be made through an hypodermic needle, and the contents placed in an open basin. The high lipoid content will frequently impart a shimmer or yellow sheen to the fluid. It is true, however, that cholesterol crystals are not always present in branchial cysts, especially if such cysts have been the seat of repeated active inflammation. Thus we see that examination of the aspirated contents is not infrequently without special value, but does not entirely eliminate a cyst.

Wangensteen has recently added to our armamentarium, especially in those cases where no cholesterol crystals are found, and where the diagnosis is still in doubt. After aspiration of some of the material, an opaque substance is injected, and with the needle left in situ, an *x*-ray picture is taken, after which the opaque material is reaspirated. When the condition was that of a branchial cyst, he obtained a sharply circumscribed limitation of the medium on the *x*-ray film. In the presence of a suppurative tuberculous gland or a "cold abscess" from a tuberculous spondylitis, the opaque medium would be seen to dissect along the natural tissue planes in an irregular fashion.

Thus given an usual swelling of the neck in which the diagnosis is wanting, we may be able by the history, physical findings, examination of the aspirated material, and *x*-ray evidence determine with much certainty a branchial cyst. Have we now at our command some means whereby we can with more accuracy make the diagnosis of carotid body tumor?

A method which indeed is not new, but which perhaps has not been given full credence can be used here to great advantage and will render the diagnosis almost

without exception. We refer to the biopsy by needle puncture and aspiration. Thirty years ago or so, Mixter described a punch which is generally employed to obtain brain specimens. Ward in 1912 suggested needle puncture and aspiration of lymph nodes in the study of lymphoblastomas. Guthrie in 1921 reported his observations on aspiration of nodes in Hodgkin's disease. Forkner in 1927 presented a method by which he obtained a small amount of tissue by the use of a dental broach inserted through an 18-gauge needle, attached to a Record syringe. Martin and Ellis in 1930 reported on 65 cases of neoplastic diseases in which the diagnosis was made by this method. Various types of tumors were subjected to this study, approximately one-half being done on neck nodes or masses. By this method a number 18-gauge needle attached to a Record syringe is inserted and advanced slowly through the superficial tissues until the point of the needle is felt to enter the suspected neoplastic mass. When the point of the needle is felt to enter the tumor, the piston is partly withdrawn so as to create a vacuum and by this means a plug of tissue is forced and drawn into the needle, which can be examined at once or fixed for later microscopic examination.

Hoffman, recognizing that the objection to this procedure was the possible contamination by tumor cells of the track as the needle was withdrawn, devised an 18-gauge outer insulated steel sheath which by connection to a high-frequency current coagulated not only the needle track but devitalized any adherent tumor cells, thus preventing implantation of tumor tissue along the course of the withdrawn needle.

Inserting such a needle will be of threefold value. Should the condition be that of a branchial cyst, the aspirated material can immediately be examined for cholesterol crystals, thus establishing the diagnosis almost at once. Finding no cholesterol crystals, with the needle still in place, the opaque medium can be introduced as suggested by Wangensteen, and an *x*-ray taken. The contour of the opaque material will substantiate the diagnosis of branchial cyst or suppurative tuberculous gland. The absence of any liquid content, showing the tumor to be solid, the aspirated tissue can rapidly be examined and its specific identity established, no matter what type of tumor it may be. There is little or no danger in inserting such a needle into a malignant growth, nor would any complications arise should the needle enter the carotid vessel, or, even an aneurysm in this location.

SUMMARY

Although relatively uncommon, a tumor of the carotid body must be entertained whenever a firm swelling located at the bifurcation of the carotid vessels, and extending beyond the anterior border of the sternomastoid muscle is encountered. Branchiogenetic cysts, while more common, may occupy the same anatomical position, and may present signs and symptoms indistinguishable from a tumor of the carotid body. Our present criteria in arriving at a diagnosis of tumor of the carotid body are: (1) position of the tumor, (2) its movement laterally but absent vertical mobility, and (3) its transmitted but nonexpansile pulsation. Because of the proximity and frequent attachment to the underlying great vessels of the neck, branchiogenetic cysts may fulfill all these criteria. Heretofore, the diagnosis of carotid body tumor as well as branchiogenetic cysts has been made with difficulty, and most frequently the true condition was not established until the neck had been opened, when the exact nature of the lesion became apparent. The ability to make a correct preoperative diagnosis is of utmost importance because of the tremendous inequality in the surgical difficulties encountered in the two lesions, the branchiogenetic cysts being usually relatively simple, while the carotid body tumor requires the surgical ability of a master. The mortality and morbidity in the two conditions varies markedly, in the former

being practically nil while in the latter extremely high, so that any addition or refinement in our diagnostic acumen which will permit us to differentiate these lesions should be a most welcome addition.

The history and physical findings in the two conditions may be insufficient to determine the exact status of the lesion. The introduction of an 18-gauge needle will help greatly in arriving at the proper diagnosis. By this means we may aspirate the contents, if the tumor be cystic, and by demonstrating the presence of cholesterol crystals, a diagnosis of branchial cyst is established. Should the diagnosis still be in doubt an opaque medium can be introduced through the needle and an *x*-ray film taken which will be of further aid. If, however, the tumor is discovered to be solid, a syringe can be attached to the needle, some of the material aspirated and immediately examined, thus establishing its specific identity. Should the lesion be an aneurysm or should the needle enter any of the large vessels no damage can result.

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VASCULAR OBSTRUCTION OF THE URETER IN JUVENILES*

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ALTHOUGH upper ureter obstruction by anomalous renal vessels is a frequent finding in adults, in many of whom the urologic symptomatology dates from early childhood, rarely has the lesion been clinically identified in infants and children. The clinical discovery of vascular obstruction of the lower ureter has been recorded in but one adult,[1] never in an infant. By urologic examination of five children with chronic pyuria and two others in whom nephritic hematuria and renal pain were the cystoscopic indications, the diagnosis of aberrant blood vessel obstruction of the ureter was made and later confirmed at operation. In an additional case of mild persistent pyuria not operated upon, the diagnosis of vascular obstruction seems the likely one. An eighth case, an autopsy finding, is also reported. Two cases of this series are unique. The first (Case II) because, as far as I can determine from a careful study of the literature, it is the youngest patient in whom this type of upper ureter obstruction was correctly diagnosed preoperatively. The other unusual case (VII), vascular obstruction of the lower ureter, is the only one recorded in an infant and is the second of its kind to have been clinically identified.

Because obstructive lesions of the ureter produce renal back-pressure and urinary stasis they assume great importance. Back-pressure causes hydronephrotic destruction with coincident function loss. Stasis predisposes to and perpetuates urinary infection which is manifested by pyuria and in children is commonly misdiagnosed chronic pyelitis. In these cases the usual attempts at medical therapy are made; the disease progresses. Ultimately nephrectomy for hydropyonephrosis is usually demanded. Only in rare instances are these young patients given the benefit of a complete urological examination without which accurate diagnosis and rational therapy are impossible. A complete urological examination should be carried out in all children in whom pyuria persists after four weeks of intensive medical therapy.[2]

Anomalous renal vessels are so common that no operator experienced in renal surgery mobilizes the kidney without meticulous care that fibrous bands passing towards the spine are cut only after determining them to be free of ectopic vessels. If doubt exists ligature is liberally employed. In an examination of 1237 kidneys, Eisendrath and Straus[3] reported accessory vessels in 21 per cent. Anomalous vessels traversing the ureter occur in 2 per cent of all individuals and are a potential cause of ureteral compression. They may arise from the aorta, vena cava or renal pedicle. In one in 400 individuals these vessels arise from the common iliacs. In two-thirds of all cases of vascular obstruction the renal vessel passes anterior to the ureter. As Jeidell has shown,[4] these vascular anomalies are due to the retention of blood vessel attachments attained before, but normally lost after, "ascent" of the metanephros from its fetal pelvic position.

Among urologists there is lack of agreement upon the etiologic relation of aberrant vessels to hydronephrosis. The minority believe that hydronephrosis results primarily from vascular ureteral compression. The majority, whose views have been adequately expressed by Albarran, Mathé and others, believe the obstructive lesion

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is secondary to renal ptosis. Statistical autopsy and surgical data indicate the extremely high incidence of disease-free kidneys which are normal in position yet show anomalous lower polar vessels.

Fig. 1. (Case 1.) Massive hydronephrosis due to aberrant vessel obstruction in a five-year-old girl. There was marked infection and no function. Nephrectomy was performed with cure of "chronic pyelitis."

With primary renal ptosis the ureter comes to hang or sag over a fixed point: the blood vessel or, occasionally, a periureteral band, and with development of hydronephrosis and further ptosis, ureteral compression increases. A vicious cycle is thus established. Factors predisposing to ptosis are: a shallow lumbar gutter, lax perirenal fascial or peritoneal attachments, scant perirenal fat, generalized visceroptosis or repeated renal trauma such as jumping, jarring and so forth.

Antecedent acute renal infection with peripyelitis and periureteritis and the subsequent development of fibrous adhesions about the ureter or even stricture formation conceivably may predispose to the initial hydronephrotic change. Commonly, however, congenital ureteropelvic junction stricture causes the primary hydronephrosis. Later, renal sag with ureteral curvature and compression over the fixed point (vessels or fibrous band) occurs. Neither the clinical course nor the pathological findings assist greatly in the determination of the initial etiologic factor. Quinby has suggested that ureterovascular contact with continuous arterial beat irritation suffices to disrupt or inhibit the normal ureteral peristalsis and thus incites pyelectasis.

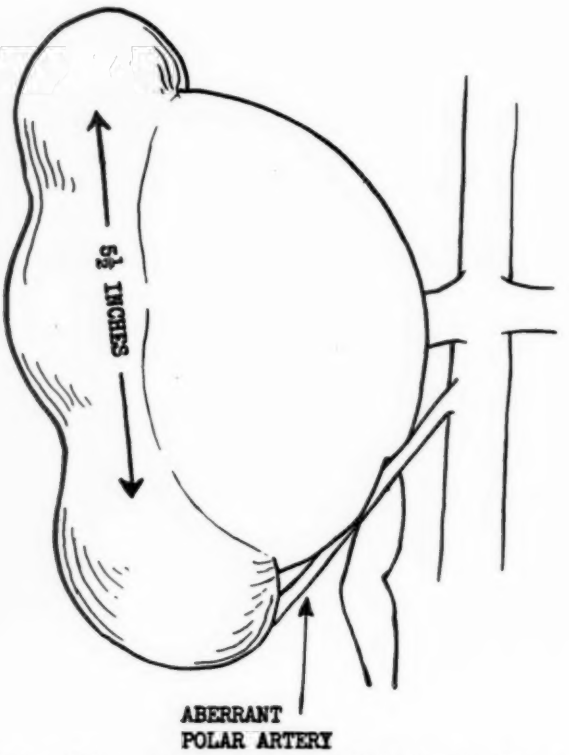


Fig. 2. Schema of vascular ureteral blockage in Case 1. A similar finding was observed at autopsy in Case viii.

Unquestionably in certain instances the hydronephrosis is induced by lower ureteral obstruction particularly stricture. By ureteral dilatation this channel may be secondarily compressed against an anomalous vessel and dilatation above this point is thereby increased (Figs. 1, 5, 10). In three infants the urogram indicated ure-

terovesical junction stricture with ureteral dilatation and secondary compression against a vessel traversing the lower

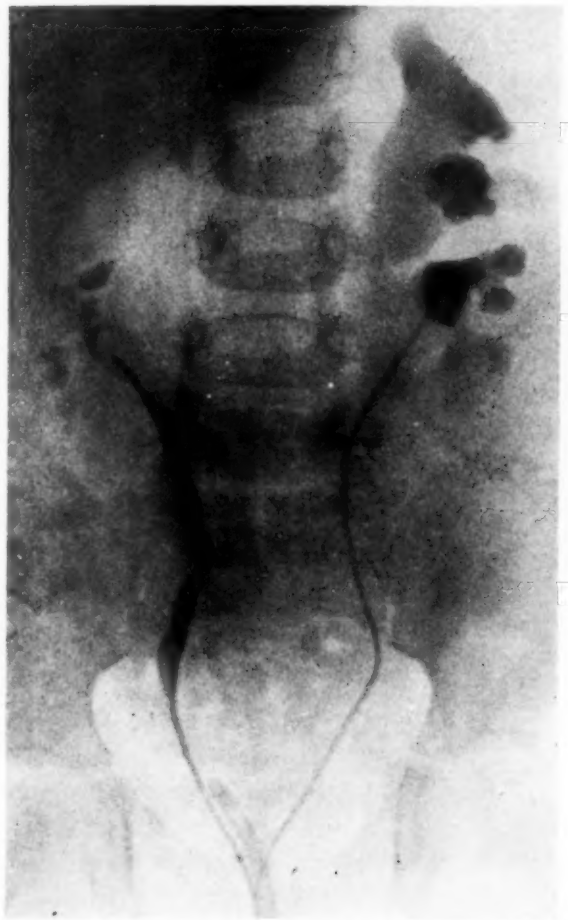


Fig. 3. (Case II.) Hydronephrosis in two-year-old girl treated eighteen months for so-called "chronic pyelitis." At operation enormous bifid pelvis was found to be chiefly extrarenal. Because entrance of ureter catheter in pelvis was followed by a steady hydronephrotic drip and ureter failed to fill on urographic injection, preoperative diagnosis was ureteropelvic obstruction probably due to an aberrant vessel. Nephrectomy was performed because of far-advanced parenchymal destruction. Recovery. Remaining kidney is of good function, uninfected and urographically normal.

ureter. But one of these patients came to operation and is therefore, the only one included in this series. An artery, vein and fibrous band were found firmly compressing the ureter 1.5 cm. above the ureterovesical junction (Case VII, Fig. 13).

Anomalous vessels obstructing the lower ureter in the region of the bladder are usually derivations of the hypogastric artery or vein or of their umbilical branches. The observations of Hyams[1] indicate that

A B

Fig. 4. Left kidney removed in Case II. Courses of multiple anomalous renal vessels are indicated by threads attached to ligated vessels, lowermost of which, an artery and a vein, caused essential blockage. At time of operation, pelvis was greatly distended; infundibulum did not exist but appeared after relief of intrapelvic pressure. Parenchyma was extremely thin, flabby and universally infected. A. Anterior view. B. Posterior view. Noteworthy is anomalous vessel passing to perirenal fat from lower pole convexity.

the umbilical and the inferior vesical vessels (the latter are derived from the umbilicals or directly from the hypogastrics) are most likely to cause ureter obstruction. Because the venous usually accompanies the arterial supply, when vascular ureter obstruction exists, both artery and vein should be sought; frequently only one will be found.

RENAL PATHOLOGY

The renal changes are those common to severe urinary back-pressure. Infection ultimately occurs to intensify and accelerate the destructive process. The hydronephrotic pelvis may be ten times normal size. By intrarenal compression and consequent vascular disturbances, the paren-

chyma is often reduced to a thin shell (Fig. 11). Marked hydronephrosis with thinned weakened walls renders these

Fig. 5. Pyelogram in Case III. There is an enormous hydronephrosis sharply cut off at ureteropelvic junction; no ureter filling. Right pelvis is normal.

organs particularly liable to rupture with only moderate trauma, as we have twice observed in young adults. With renal infection, fibrolipomatous perinephritis is common.

Microscopically the kidney shows tubular dilatation with loss of epithelium, more or less glomerular destruction, and widespread interstitial sclerosis with localized or diffusely scattered areas of infection or suppuration. In far advanced lesions the medulla and cortex can be differentiated with difficulty or not at all.

Resultant renal function loss is of prime clinical concern; only slightly less important is bacterial toxic absorption as manifested by systemic symptoms or by toxic injury of the other kidney. In one of our patients (Case VI) the clinical picture was that of chronic interstitial nephritis with hematuria. In another (Case V), toxic

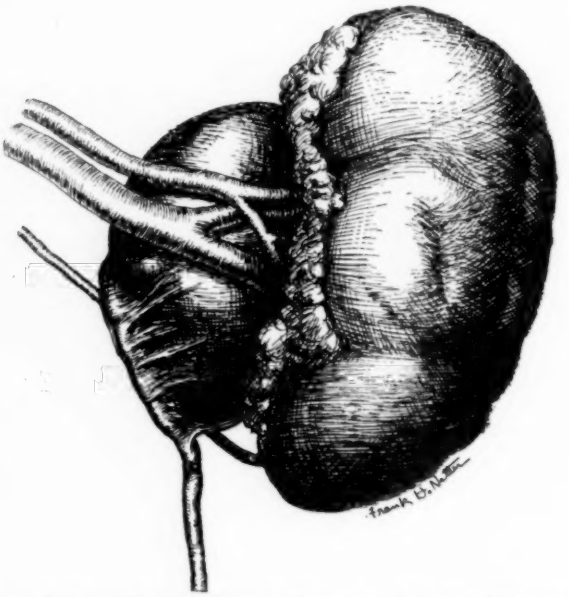


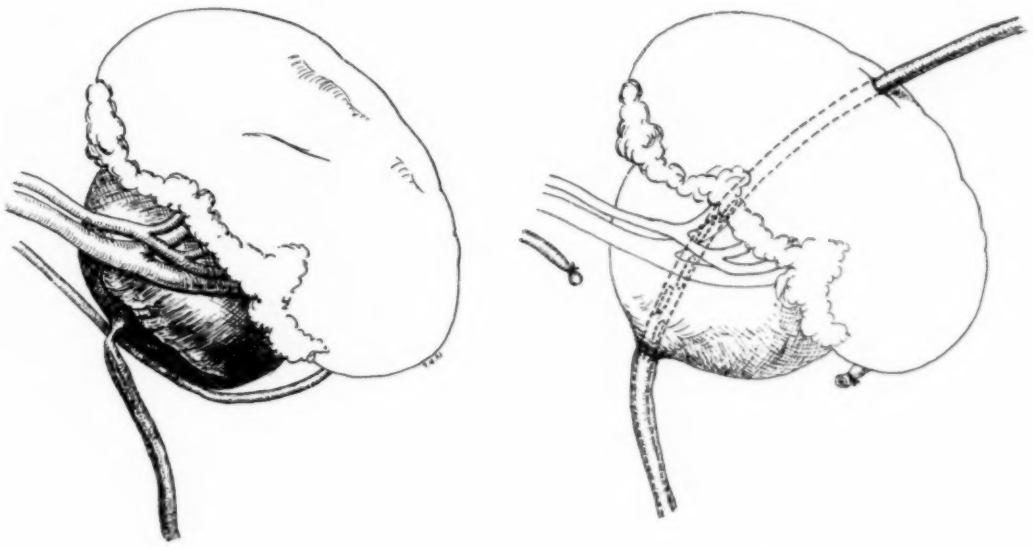
Fig. 6. Operative findings in Case III. (Fig. 5.) Kidney was somewhat ptosed, ureteropelvic junction elevated and ureter bound to pelvis with dense fibrous bands. Aberrant lower polar vessel is shown.

nephritis of the better kidney was severe and, to digress, indicates a possible etiology in certain cases of so-called Bright's disease. Fortunately, removal or adequate conservative treatment of the diseased kidney is often followed by complete disappearance of the toxic nephritis in the good organ and the assumption of satisfactory function.

SYMPTOMS

The symptoms of ureteral vascular obstruction are predominantly those of hydronephrosis, usually infected, and are frequently characterized by intermittency. As indicated in the initial paragraph, these symptoms often begin in early childhood. In two adults recently operated upon for aberrant vessel hydronephrosis, both reported that from earliest childhood they found that the dull loin ache (of hydronephrosis) temporarily could be relieved by firm pressure with the fist or by pushing

the loin against a piano or door. Thus they managed forcefully to evacuate the hydronephrotic sac so that distention pain did not recur until urinary secretion again overdistended the pelvis.



A B

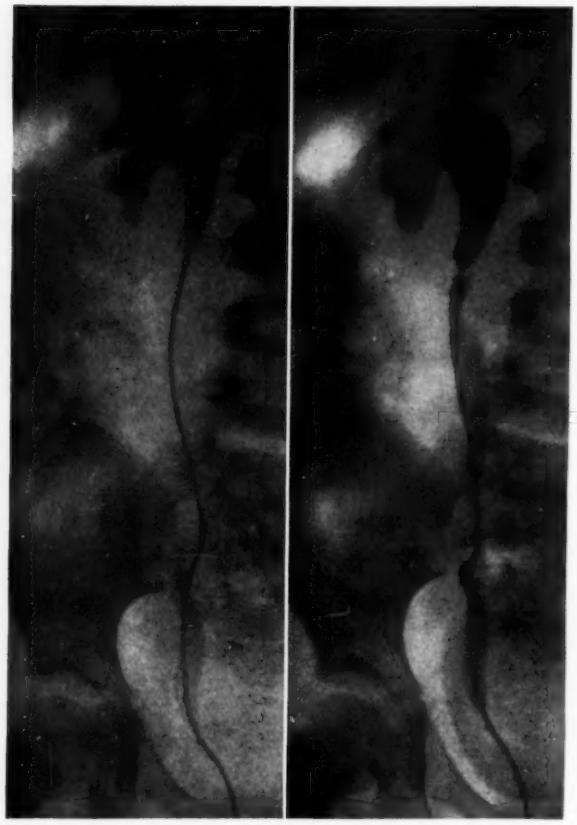
Fig. 7. (Case III). A. Appearance following dissection of ureter from pelvis (see Fig. 6) showing ureteropelvic junction stricture. A Heinecke principle plastic operation was attempted but was unsuccessful; ureteral atresia was too marked. B. Treatment in Case III. Ureteropyeloneostomy with nephrostomy drainage. No. 10 F. ureter catheter was passed 10 cm. into ureter for drainage and splinting. Several large drainage holes were made in catheter where it traversed pelvis. Additional pelvic drainage was offered by a pyelostomy tube (not shown). Infection required secondary nephrectomy.

The symptoms may be divided into (1) those referable to the urinary tract and (2) general.

1. A dull loin ache, sometimes punctuated with renal colics, with or without radiation, is perhaps the most common subjective symptom; it is one for which relief is often sought. In one of our patients (Case 1), at the age of three years appendectomy had been performed during an acute recurrent attack of colic with abdominal pain, fever and vomiting. Renal enlargement is often noted by the child or mother; its periodicity is diagnostically valuable. In the absence of infection, urinary frequency may be reflexly produced. With urinary infection frequency, burning, dysuria and hematuria are common but to the parent and physician persistent pyuria may be the most distressing observation. Five of the 7 clinical cases here reported were referred for examination because of "chronic pyelitis." With acute obstruction the pyuria may disappear completely but the simultaneous sudden onset of severe symptoms of renal infection with localizing signs should always suggest the course of clinical events.

2. Systemic symptoms are those referable to diminished renal function and toxic absorption. As long as the remaining kidney compensates adequately, the total renal function loss will be evident only by laboratory estimation. With an unreliable renal mate (congenitally or surgically absent, hypoplastic or injured by toxemia) sub-uremic or uremic symptoms appear: headache, irritability, malaise, marked gastrointestinal upsets or even stupor. With infection, an intermittent low grade fever is regularly found; gastrointestinal upsets occur in over half of all cases. Anorexia, malaise, anemia, "biliousness," dyspepsia, chronic gastritis, constipation, diarrhea, nausea or even vomiting as well as loss of weight or failure to gain

are common complaints in children with obstructive uropathy. Many children with urinary obstruction are treated months or years for stomach trouble.



A B

Fig. 8. Left pyelogram (picture reversed) in Case iv. A. With moderate pelvic filling, there is very slight reflux down ureter past site of upper ureteral blockage. B. With marked distention site of transverse ureteral obstruction becomes more evident. Improved by ureteral dilatation (no operation). We believe an extra-ureteral compression exists, most likely a vessel, and a low ureterovesical junction narrowing has secondarily rendered extra-ureteral lesion of clinical importance.

In children with the foregoing symptoms, particularly a fever of obscure etiology, a careful urinalysis should be repeatedly made. If pyuria persists longer than one month despite intensive medical therapy (eradication of focal infections, forced intestinal elimination, nutritional improvement and the liberal employment of urinary antiseptics) a complete urological examination should be performed. Only by this method can the correct diagnosis be made when the lesion is a soft tissue obstruction of the upper urinary tract.

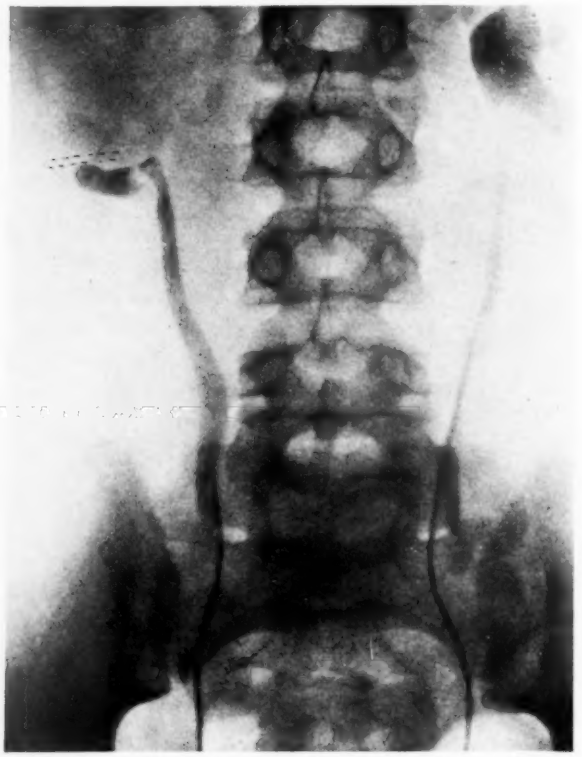


Fig. 9. (Case v). Aberrant vessel obstruction in eleven-year-old male which caused total renal destruction. Clinical findings were those commonly observed in chronic interstitial nephritis. Course of vessel is indicated. Nephrectomy. Right pyelogram (picture reversed) normal.

Routinely we acquire the following precystoscopic data: urinalysis (particularly pus, blood and bacteria), blood urea content and CO_2 combining power, a two-hour phenolsulphonphthalein output estimation, a plain roentgenogram of the urinary tract for calculi and spinal defects, a cystogram and an intravenous urogram. Frequently, the preliminary findings are of great value for the more comprehensive performance of cystoscopy and retrograde pyelography. With these data in hand cystoscopy, ureteral catheterization, divided renal function tests with pyelography on the indicated side are carried out. Occasionally the ureter catheter will not pass the obstruction (Case v). Pyuria and diminished function

are quite regularly found on the diseased side. The pyelogram shows an abrupt transverse ureteral obstruction with hydronephrosis above; sometimes the latter is extreme. The vascular course is sometimes indicated as a filling defect in the pyelographic medium (Figs. 1, 8B, 13A) or may completely withhold the media above (Figs. 5, 8A). A large hydronephrosis sharply demarcated at the ureteropelvic junction is always suggestive though not pathognomonic of aberrant vessel obstruction. Aberrant vessel, localized periureteropelvic sclerosis, tight ureteral stricture or an abnormally high ureteropelvic insertion with valve formation may produce the same pyelogram. However, all of these conditions demand surgical exploration. Parenthetically, it should be added that in hydronephrosis, urographic injection is often followed by acute edematous obstruction and should not be performed unless one is prepared to operate at once, if necessary.

Fig. 10. Nephrectomy specimen in Case v. Enormous pelvis is now collapsed. Course of anomalous vessel obstructing ureter is admirably shown. Parenchyma looks sound and ample but is only a moderately thin shell (see Fig. 11).

Fig. 11. Histosection of entire kidney shown in Figure 10. Noteworthy is pelvic dilatation with calyceal separation and great thinning of parenchyma. At upper pole a portion of parenchyma approximates half normal thickness.

In the lower ureter a transverse filling defect (Fig. 13) may be due to vascular or periureteral fibrous obstruction, plication or congenital valves of the ureters. Absence of similar urographic findings in the dilated tract above should rule out plication and valves. We have had but one case of urographically demonstrated congenital ureteral valves; the finding was confirmed at autopsy. The ureterographic appearance suggested a string of short thick sausages.

TREATMENT

In vascular obstruction, so-called conservative treatment: kidney belts, ureteral dilatation, pelvic lavage or strictly medical therapy, serve only to emphasize the necessity of operative attack. On the other hand, when possible, conservative surgical treatment should be liberally employed. The renal arterial circulation is terminal; severance of an artery means function loss of the tissue thereby supplied. Moreover, vascular section is sometimes followed by parenchymal necrosis and suppuration.

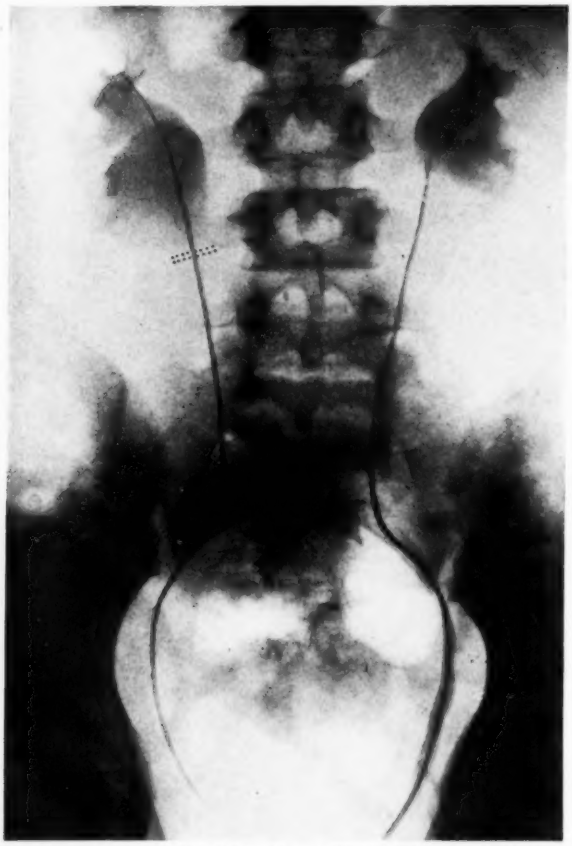
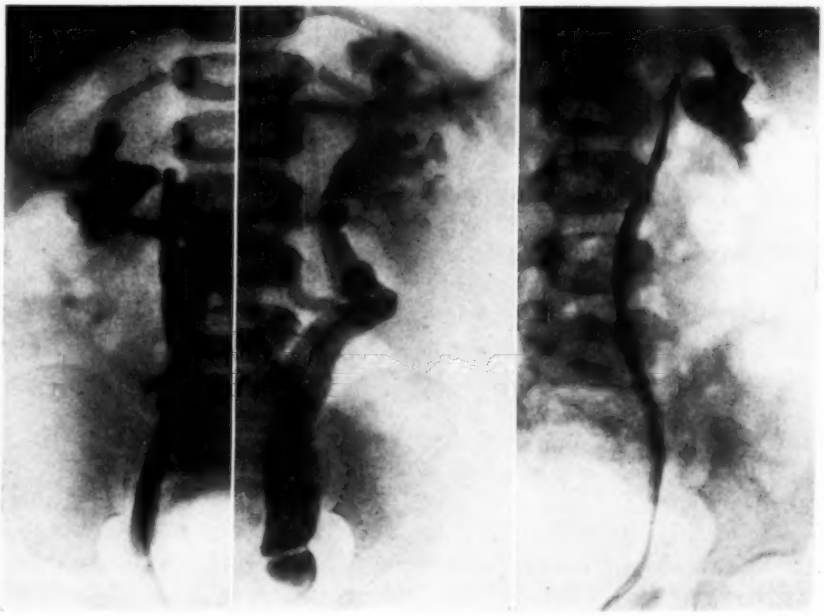


FIG. 12. Case VI. Left hydronephrosis due to ureteropelvic stricture and aberrant vessel obstruction (picture reversed). Right pelvis is unusually large but finer calyceal markings are well preserved. In this patient, a girl of twelve years presenting clinical picture of advanced interstitial nephritis, left aberrant resection with Heinecke principle ureteroplasty resulted in striking clinical improvement. Renal biopsy showed very mild interstitial nephritis (see case report).

Unless the amount of the parenchyma supplied by the aberrant artery is small, certainly less than a fourth of the organ, ureteropyeloneostomy (Figure 7, B) or some other form of ureteropelvoplasty to circumvent the obstructing vessels should be performed. Young[5] has recently described a pyeloplasty technique to accomplish this. The kidney is delivered, the ureter is mobilized and determined to be free of stricture or periureteral adhesions. From the anterior pelvic wall an elliptical area is excised and the opening is closed so as to draw upward and inward that portion of the pelvis previously in contact with the obstructing vessels. Following a similar though more radical posterior pelvic resection, by closure of the defect, the ureteropelvic junction is drawn upward and backward to separate widely the ureter from the obstructing vessels. Ureteral stricture at the point of vascular crossing sometimes may be eradicated by utilization of the Heinicke-Mikulicz principle: longitudinal incision through the stricture with transverse suture. We did this in one child (Case VI); the result was functionally but not anatomically satisfactory.

In all conservative attacks on obstructing aberrant vessels, it is essential that the lower portion of the pelvis and upper ureter be well mobilized, straightened out and periureteral bands or adhesions freed and cut lest in renal replacement an obstructive kink persist. Many believe that complete mobilization alone is needed. Following kidney replacement, nephropexy is recommended to assist in the maintenance of a high renal position and dependent drainage. Our preference is the peritoneo-fascial hammock method of Deming with which no sutures penetrate the kidney.[6] Periodic postoperative follow-up examinations are essential to determine definitely that satisfactory renal drainage has been effected. Subsequent unfavorable functional or symptomatic progress demands nephrectomy when the condition of the renal mate permits.

In some instances the hydronephrotic kidney is totally destroyed. Because of this nephrectomy was required in 4 of



A B C

Fig. 13. A. and B. Composite urogram in a female of thirteen months (Case VII) examined because of persistent pyuria. Bilateral ureterovesical junction stricture with badly infected left ureterohydronephrosis and almost complete function loss. On right side ureteropelvic dilatation is less marked but infection and functional diminution existed. Right ureteropelvic junction is peculiarly angulated, probably due to ptosis. Lower right ureter consistently showed a complete transverse filling defect which we interpreted as due to vascular obstruction or a periureteral band, probably secondarily operating to cause blockage because dilated ureter became compressed against it. Left ureteronephrectomy. Condition of patient did not warrant investigation of left ureter or bladder. Subsequent exploration of right lower ureter revealed a transverse blockage by artery, vein and fibrous band. Following section of these, local ureteral filling promptly occurred. C. Right pyelogram (picture reversed) one year later. Transverse filling defect is now absent; upper tract dilatation is diminished; renal ptosis persists (see case report).

our patients. Associated calculous disease or widespread infection usually indicates nephrectomy. Unless the integrity of the opposite kidney has been established preoperatively, nephrostomy with a large tube drainage usually will safely tide the patient over until the function of the better organ can be determined. In one of our patients (Case v), operated upon five years before in another hospital without preoperative urologic study, this method was advantageously followed. While the acute massive hydronephrosis was temporarily relieved, failure to recognize and treat the anomalous vessels as the underlying cause of the renal lesion, resulted in subsequent total destruction and loss of the kidney.

CASE REPORTS

Case I. R. D., female, aged five years, was admitted to the Children's Medical Service of Bellevue Hospital November 29, 1927. The chief complaint was repeated spells of vomiting during the past three years. These vomiting episodes lasted two or three days at a time and recurred every four to six weeks. There was also fever, pain in the lower abdomen and coffee-colored urine. Appendectomy was performed at the age of three years because of and during one of these attacks of abdominal pain and vomiting. For six weeks previous to admission the abdominal pain, particularly in the right loin, was severe; urinary urgency existed and terminal spasm accompanied each urination. During this period the temperature showed a daily rise of about 3°.

Physical examination revealed a well-nourished and well-developed child who did not look sick. Neither kidney could be palpated. There was a marked pyuria with moderate albuminuria. No red blood cells or casts were

found. The blood non-protein-nitrogen was 35 mg. per 100 c.c.; the blood pressure 110/70. Two-hour phenolsulphonphthalein test intramuscularly showed 20 per cent return in the first hour and 20 per cent in the second hour.

Urologic Examination: The cystogram was normal. Cystoscopy without anesthesia revealed a turbid bladder urine and a moderate generalized cystitis with some edema of the trigone. Catheters were passed 18 cm. to the kidney pelves without difficulty. From the left side a deep amber, sparkling urine with good flow was obtained. From the right side the catheter return was pale, watery, grossly purulent and less in quantity than that of the left. Two cubic centimeters of 0.5 per cent indigocarmine injected intravenously appeared after five minutes in good concentration from the left side. None returned from the right side in fifteen minutes' observation. Right pyelography was performed; 10 c.c. of 12 per cent sodium iodide were painlessly injected. Two cubic centimeters of iodide were injected on the left side to outline faintly the left pelvis.

The *pyelogram* is shown in Figure 1. Enormous hydronephrosis is evident and a broad clear band devoid of iodide shadow is seen passing obliquely upward and across the region of the ureteropelvic junction (Fig. 2). A diagnosis of aberrant renal vessel obstruction was made and operation advised. The ureterogram suggests that ureterovesical junction stricture was probably the primary obstruction.

Operation, January 17, 1928. Right loin kidney exposure revealed this organ to be twice normal size and lobulated. At the lower pole a firm nodule of parenchyma was found but elsewhere the kidney was extremely thin, sacculated and infected. A large extrarenal pelvis existed and an aberrant vein passed obliquely from the lower renal pole upward to the vena cava firmly blocking the ureteropelvic junction as indicated in the pyelogram and diagram (Fig. 2). Immediately below this aberrant vessel the ureter was somewhat kinked and surrounded by fibrous bands which may have been primary or secondary obstructive factors. Because of extreme parenchymal destruction, nephrectomy was performed. Convalescence was uneventful. Phenolsulphonphthalein test ten days postoperatively showed 55 per cent excretion in two hours. Scattered pus cells still were found in urine but unquestionably were due to persistent ureteritis and cystitis. When seen six weeks postoperatively the patient had no complaints, the urine contained no albumin and was microscopically normal.

In addition to the interesting anatomic lesion in this case certain phases of renal function are noteworthy. It would seem that the albuminuria noted preoperatively resulted chiefly from toxic nephritis of the good kidney. That this is likely is indicated by the marked improvement in renal function following the removal of the badly infected hydronephrotic mate. The latter performed no work and was a toxic source which not only injured the good organ but increased its functional burden.

Case II. N. B., female, aged two years, was referred by Dr. W. M. Winters of Paterson, N. J. for urologic examination because of chronic pyuria of eighteen months' duration. Medical therapy had been exhaustively administered.

Past history was negative except for periodic febrile attacks and gastrointestinal upsets. During recent weeks the child had indicated pain in the left loin.

Physical examination revealed a well-developed, well-nourished baby with a slightly enlarged and tender left kidney.

Urologic Examination: Urinalysis: cloudy, acid, sp. gr. 1.014, albumin +, sugar 0, pus ++++, R.B.C. occasional, culture B. coli, non-protein-nitrogen estimation showed 27.2 mg. per 100 c.c. of blood. Phenolsulphonphthalein specimens were lost by voiding. A plain urinary tract roentgenogram and cystogram were normal.

Cystoscopy: Gas-oxygen anesthesia. The bladder urine was grossly purulent. There was a mild generalized cystitis with edema about the left ureteral orifice. Both ureters were catheterized easily, a steady drip suggestive of hydronephrosis was obtained from the left pelvis. The right ureteral return was amber, clear and sparkling. The left urine was watery and cloudy; 40 c.c. were aspirated from the left pelvis. Indigocarmine intravenously appeared in deep concentration from the right catheter in three minutes and extremely faintly from the left side after twelve minutes. Laboratory examination of these specimens showed a

normal sterile right urine; that from the left was watery, heavily pus laden, of low urea content and infected with B. coli.

A bilateral *pyelogram* shows marked left hydronephrosis with no filling of the ureter below the kidney pelvis (Fig. 3). The right urogram is normal. Preoperative diagnosis: probably aberrant vessel obstruction with infected hydronephrosis.

Operation, November 11, 1931. Left nephrectomy. The organ was four times normal size and an enormous extrarenal pelvis with lower pole anomalous vascular obstruction (Fig. 4) existed. The renal vascular supply was thoroughly anomalous and consisted of a series of vessels passing to the kidney. In addition, a large vessel from the perirenal fat entered the convexity of the lower pole (Fig. 4, B). The parenchyma was a thin, soft, flabby, badly infected shell which was so diseased that removal was deemed wisest.

Convalescence was satisfactory with discharge from the hospital on the thirteenth day. The urine at this time still showed an occasional leucocyte. The phenolsulphonphthalein output was 52 per cent in two hours. Subsequent urinalyses have been normal.

Apart from the demonstrated relation of congenital vascular obstruction to so-called chronic pyelitis with renal destruction, this patient is of unique interest because she is the youngest reported in whom the preoperative diagnosis of aberrant vessel obstruction was correctly made. Fister and Smith record this operative findings in an infant of twenty-two months in whom the lesion was unsuspected preoperatively.[7]

Case III. R. P., female, aged eight years, on November 11, 1931 was admitted to the Children's Medical Service of Bellevue Hospital with a history of periodic attacks of left kidney pain accompanied by fever and vomiting. The attacks had recurred every two to four weeks; the pain was severe but did not radiate. Such an attack had existed for twenty-four hours previous to hospital admission and was the first in two months.

Physical examination was essentially negative except for some left renal and costovertebral angle tenderness without palpable kidney enlargement.

Urologic examination: Urinalysis: Cloudy, sp. gr. 1.014, albumin +, sugar 0, W.B.C. +++, B. coli and Staphylococcus albus. The non-protein-nitrogen estimation was 34 mg. per 100 c.c. of blood; phenolsulphonphthalein (intramuscularly) first hour 35 per cent; second hour 20 per cent. A plain roentgenogram of the urinary tract was negative. A cystogram showed a large bladder.

Cystoscopy, November 19, 1931: No anesthesia. Bladder urine was clear and sparkling; the bladder picture was normal. Both ureters were catheterized easily. Indigocarmine intravenously returned in deep concentration from the right side in ten minutes; none returned from the left ureter in one and one-half hours. A bilateral pyelogram was made but showed no left pelvic outline; the media did not go above the ureteropelvic junction. Ureteral catheterization was subsequently repeated at which time the ureter specimens showed:

	Pus	*Urea*	*Culture*
Right kidney.	Negative	.01 per c.c.	Sterile
Left kidney...	5–8 h.p.f.	.002 per c.c.	B. coli and Staph. albus
Bladder.....	.....	.....	B. coli and Staph. albus

The *pyelogram* showed a normal right pelvis with an enormous left hydronephrosis sharply demarcated at the ureteropelvic junction (Figs. 5, 6, 7). A diagnosis of aberrant vessel obstruction was made and because of some remaining renal function, a conservative ureteropelvoplasty was advised.

Operation: This was done December 4, 1931 as shown in Figure 7. The parenchyma felt solid and quite thick. At first a Heinecke plasty was attempted at the site of ureteropelvic stricture but was unsatisfactory so that ureteropyeloneostomy was done, a large (10 F.) nephrostomy-transpelvo-ureteral catheter being left in place for ureteral splinting and pelvic drainage. Pyelotomy drainage was also afforded by a No. 24 F. mushroom tube.

The postoperative course was exceedingly stormy with extensive fascial sloughing in forty-eight hours. The renal tube drainage was technically satisfactory but extremely purulent. Four transfusions did not help. The infected wound was twice opened widely. Secondary

nephrectomy was performed December 19, 1931 with an uneventful though prolonged convalescence. The patient was discharged healed February 22, 1932 with a normal urine.

The removed organ resembled the so-called "large white" kidney of nephritis but microscopically showed widespread suppurative pyelonephritis.

In this patient infection defeated conservative ureteroplastic methods.

Case iv. A. F., female, four years, was examined May 16, 1930 because of persistent pyuria of two months' duration. The child was anemic but otherwise appeared normal. Neither kidney was felt; there was no loin tenderness.

Urologic Exanimation: The urine was slightly hazy, contained no albumin or sugar but it showed a moderate number of leucocytes (15–40 h. p. f.) and on culture, B. coli. A plain roentgenogram and cystogram were normal. Ureteral catheterization without anesthesia returned slightly hazy urine from the left side and contained about as many pus cells as the bladder urine. Right catheter urine was normal with 5 gm. of urea per liter in contrast to 4 gm. from the left side. The indigocarmine output following intravenous injection was equal and normal on each side. The left pyelogram (picture reversed) (Fig. 8) indicates, to the writer, polar vessel obstruction (or perhaps a periureteral fibrous band) which may be secondarily manifested because of pressure of the dilated ureter against the extraureteral object. Although the urographically apparent stenosis of the ureterovesical junction suggested this to be the initial site of blockage, four No. 4 f. catheters were passed to the renal pelvis side by side without the slightest difficulty. On catheter withdrawal the bladder wall was not pulled along as we are accustomed to observe with ureterovesical junction stricture. Following this catheter dilatation the pyuria disappeared; the child was not operated upon. Follow up urogram showed the same picture as at first. This case is included here because of its illustrative urography and because it is believed to fall in the group under consideration.

Case v. J. D., male, eleven years, was examined March 14, 1931 because of the following history. Six years previously he developed an acute left hydronephrosis with hematuria but no pyuria. A left lower pneumonia was said to coexist. Nephrostomy drainage in another hospital returned sterile urine and was followed by an uneventful recovery. Periodically since then, about four times a year, there were attacks of sharp left renal pain with hematuria lasting four to seven days. A large mass would appear in the left loin. Most of these attacks were afebrile. The last attack, four months previous to the present examination, was the most severe. Pain and fever confined the boy to bed for three weeks.

Physical examination revealed a well-developed, well-nourished boy with a left nephrostomy scar. Neither kidney could be felt.

Urologic Examination: Urinalysis showed a clear urine containing a marked trace of albumin, no sugar, scattered red and white blood cells with many hyalin and granular casts. Blood non-protein nitrogen showed 33 mg. per 100 c.c.; the phenolsulphonphthalein output was 35 per cent in two hours. A plain roentgenogram showed a large left renal shadow.

Cystoscopy under local anesthesia showed a normal bladder picture. Ureteral catheterization returned a clear sparkling urine from the right side and contained scattered single white and red cells, many hyalin and granular casts, and 1.6 gm. of urea per 100 c.c. All cultures were sterile. Indigocarmine intravenously appeared in medium concentration from the right side in ten minutes, later becoming more concentrated. No dye returned from the left side in twenty minutes. Pyelogram (Fig. 9) shows a peculiar ureteropelvic obstruction. A diagnosis of aberrant vessel block was made and operation advised.

Operation: April 14, 1931. Gas-oxygen anesthesia. Left loin kidney exposure with excision of old scar disclosed the kidney to be firmly bound in a dense mass of perirenal fibrosis with almost no perirenal fat. The organ was distended; the large extrarenal pelvis was 10 cm. in diameter and was emptied by aspiration. Mobilization of the kidney revealed the obstruction: periureteral sclerosis and aberrant artery blockage at the site shown in the removed specimen (Fig. 10). Nephrectomy was performed with cigarette drainage and multiple layer wound closure. On section the organ was found reduced to a thin shell (Fig. 11). Uneventful recovery followed with discharge from the hospital twelve days after operation.

Follow up examination after two months showed no albumin, pus or casts, a rare red

cell and the phenolsulphonphthalein output had increased to 55 per cent in two hours.

Case VI. E. O., female, aged twelve years, was admitted to the Babies' Hospital May 14, 1931 because she had been told elsewhere she was "verging on nephritis."

Past History: The child had never been well and strong. She had had nocturia and frequency since birth. She had been through tonsillectomy, pneumonia, varicella, pertussis, scarlet fever (at eight years) with otitis media and myringotomy as well as frequent attacks of bronchitis.

Present History: One year after scarlet fever (at eight years) she suffered sore throat, bronchitis and fever with edema of face, hands and feet. This was followed by headaches and colds. The latter kept her in bed for a week at a time. At the time of hospital admission she had a severe, painless hematuria of three days' duration.

Physical examination showed an anemic, frail, underweight girl with pale puffy face, pretibial edema and palpable left kidney. The blood pressure was 150/80.

Urologic Examination: Urinalysis: alkaline, sp. gr. 1.011, albumin +, sugar 0, acetone 0, R.B.C. ++++ (gross hematuria), W.B.C. ++, occasional hyalin and granular cast. The nonprotein nitrogen was 48.9 mg. per 100 c.c. of blood, creatinin 1.7 mg.; chlorides 445 mg., sugar 107 mg., calcium 10 mg., phosphate 6.2 mg. The serum albumin was 4.24, serum globulin 2.79, and the CO_2 combining power 41.8 per cent. Phenolsulphonphthalein output was 12.5 per cent the first hour, 7.5 per cent the second; total 20 per cent. Tuberculin reactions were strongly positive.

Because of the findings in this case, especially the large left kidney and marked hematuria, together with personal previous observations in a few cases of apparent nephritis on an obstructive basis, a urologic examination was carried out. The results follow:

Plain urinary tract roentgenogram was negative. A cystogram (May 22, 1931) showed reflux up the left ureter to the ureteropelvic junction; no media entered left pelvis or the right ureter. Intravenous urogram (May 25, 1931) showed left hydronephrosis with evidence of ureteropelvic obstruction; the right pelvis appeared normal.

Cystoscopy (May 26, 1931): No anesthetic. Bladder urine was cloudy with a faint gross hematuria. The bladder picture was normal. Catheters were passed 20 cm. up each ureter encountering, on the left side, a moderate hydronephrosis from which several cubic centimeters of urine were aspirated. Indigocarmine intravenously appeared in seven minutes from each side. The specimen from the left side was hazy and of medium concentration; that from the right was less in amount and of slightly deeper dye concentration but definitely blood-tinged. Bilateral pyelogram showed left hydronephrosis; the right pelvis is slightly larger than usually seen in a child of this size (picture reversed) (Fig. 12). On the left side the ureteropelvic blockade was always sharply defined; the ureter below showed no filling. A diagnosis of probable vascular obstruction was made. Ureter catheter specimen examination revealed the following:

	Casts	W.B.C.	R.B.C.	*Urea*	*Culture*
Right...	Many granular	+	++++	1.2 per cent	Sterile
Left....	Many granular	+	+	.4 per cent	Sterile

Operation was advised and ultimately carried out June 29, 1931. The left kidney was exposed and delivered. The pelvis was large and chiefly extrarenal. A ureteropelvic junction atresia (stricture) existed, behind and compressing which were a large polar artery and vein. The vessels to the lower pole were ligated and cut after determining that the parenchyma supplied thereby was not extensive. The ureteral atresia was incised longitudinally and sutured transversely (Heinecke principle).

A small wedge of parenchyma was taken from the upper pole for histologic examination. Renal suspension by the Deming technique was performed. Fascia, muscle and skin were closed with cigarette drainage. An uneventful convalescence followed with discharge from the hospital on the twentieth day; a transfusion for anemia was given on the fourteenth day.

Microscopic examination (Dr. Martha Wollstein) of the removed wedge of renal tissue shows hemorrhage due to surgical trauma. The glomeruli show no changes. There is no increase in cells in the glomerular tufts, no exudate nor epithelial crescents in the capsular spaces and no fibrosis in or about any of the glomeruli. In the interstitial tissues are scattered small foci of lymphocytes and plasma cells. The blood

vessels are normal. Diagnosis: slight interstitial nephritis.

Follow-up Record: Once during the five months postoperatively, two No. 4 F. catheters were passed side by side to the left pelvis. Recent urinalyses show marked improvement. The child gained 13 pounds during the first four months postoperatively. An intravenous urogram six weeks postoperatively showed no anatomic improvement; hydronephrosis and ureteropelvic obstruction still existed.

Cases V and VI admirably illustrate the possible local etiology and pathology in certain types of nephritis. We have now 6 patients in whom congenital upper tract obstruction produced a picture clinically indistinguishable from that of advanced chronic interstitial nephritis. Renal sections in Case V are of particular interest in that the microscopic findings were in no way commensurate with the clinical observations.

Case VII. C. V., female, aged ten months, was admitted to Babies' Hospital May 21, 1929 with a history of fever and irritability of five days' duration. The onset was sudden and in addition to these symptoms there was a slight cough.

Physical examination resulted in the following diagnosis: bilateral otitis media, umbilical hernia, rickets, bronchopneumonia and pyuria. The patient was unsuccessfully treated with methenamine and discharged three weeks later. On September 4, 1929, at the age of fourteen months she was readmitted because of chronic puria with intermittent febrile attacks. At this time painful urination and increased frequency had existed for two weeks.

Physical examination on readmission in September showed injected tonsils and ear drums. There was no abdominal tenderness.

Urologic Examination: The catheterized urine specimen was milky in appearance, sp. gr. 1.015, showed a faint trace of albumin, innumerable pus cells and on culture B. coli. The blood urea was 15 mg. per 100 c.c. The phenolsulphonphthalein output was 15 per cent the first hour, 23 per cent the second, a total of 38 per cent. The hemoglobin was 55 per cent. Blood count showed leucocytes 10,400, polymorphonuclears 34 per cent and lymphocytes 66 per cent. A plain roentgenogram of the urinary tract showed no abnormal shadows. The cystogram was normal.

Cystoscopy under general anesthesia showed grossly purulent bladder urine and a marked generalized cystitis with numerous coils of mucopus on the bladder floor. Both ureteral orifices were small and tight; it was not until the third cystoscopic examination that filiform tip No. 4 F. catheters could be passed to each kidney pelvis. The ureteral return from each side showed a bilateral infection, most marked on the left side. Indigocarmine intravenously returned in moderate concentration from the right kidney in six minutes but only in faintest trace from the left side in twenty minutes. The bilateral urogram is shown in Figure 13 and indicates marked dilatation of the upper urinary tract on each side, especially the left. Noteworthy is the bilateral filling defect of the lower ends of the ureters. We made an anatomic diagnosis of congenital bilateral atresia of the ureteral vesical junction, with probable secondary bilateral vascular obstruction about 1.5 cm. above. On three occasions, therapeutic dilatation of the right ureter stricture was carried out so that eventually three No. 4 F. catheters could be passed side by side without great difficulty. This instrumentation provoked no systemic reaction. Twice, similar instrumentation of the left side provoked serious generalized reactions manifested by high fever and urinary sepsis.

Operation: In as much as the left urinary tract seemed irreparably damaged ureteronephrectomy was performed February 29, 1930. The ureter was removed nearly to its vesical junction. The feeble condition of the patient did not warrant determination of the nature of the low extra-ureteral obstruction. An uneventful convalescence ensued.

A *check-up cystoscopy* was performed May 16, 1930 (three months following left ureteronephrectomy). A catheter was easily passed to the right kidney pelvis and returned a clear sparkling urine which subsequently became hazy and on examination showed a few scattered pus cells. The pyelogram was quite identical with the original right pyelogram, still showing the transverse filling defect 1.5 cm. above the ureteral orifice. Operation for removal of the low extraureteral obstruction was advised.

Operation May 22, 1930. The patient was

twenty-two months old. Following insertion of a right ureter catheter for orientation, the lower ureter was exposed through a midline incision. At a point corresponding to that indicated in the urogram an artery and vein with a dense fibrous band were found running transversely across the ureter and firmly compressing it. Resection of this obstruction was immediately followed by normal ureteral expansion at the point of compression. The delicate condition of the child did not justify extensive dissection to ascertain the origin and destination of these vessels. It is probable that they were uterine vessels secondarily compressing the ureter already dilated by ureterovesical junction stricture; unquestionably localized periureteral fibrosis aided in the vascular obstruction.

Urinalysis ten days after operation showed a slight trace of albumin with rare pus cells. A specimen examined three weeks postoperatively showed no albumin and only scattered leucocytes and one year later the urine is normal as is the general condition and physical development.

A blood count which preceded the last operation was of interest in that it showed a reversal to normal of the polymorphonuclear and lymphocyte ratio. Total leucocytes 12,250, polymorphonuclears 73 per cent, lymphocytes 25 per cent and eosinophiles 2 per cent.

In the 6 proved clinical cases (I, II, III, V, VI, VII) of vascular obstruction, the therapeutic results have been most gratifying. Pyuria ultimately disappeared entirely in 4 cases (I, II, III, VII) in which it was an outstanding symptom. In Cases V and VI, clinically nephritic, the urinalytic evidence of this lesion disappeared in one (V) and showed marked improvement in the other. In all cases the total renal function improved.

Case VIII. A female of two years, dead of pneumonia. At autopsy, in addition to right lower lobe consolidation, an aberrant vessel obstruction of the right ureter with hydronephrosis was found (cf. Fig. 2). The urine was cloudy, apparently with desquamated epithelium; no evidence of infection was found.

SUMMARY

Seven proved cases and one probable instance of vascular ureteral obstruction in juveniles are reported. Loin pain is a frequent symptom. When infection with pyuria exists, "chronic pyelitis" is the usual diagnosis. When infection is slight or absent, the clinical diagnosis, based on urinalysis, is likely to be chronic interstitial nephritis. The existent nephritis is regularly of the toxic variety in the unobstructed organ and of the compression or backpressure type in the obstructed kidney. This nephritis may be expected to disappear following removal or adequate treatment of the seriously diseased mate. Only by complete urological examination with urography can the diagnosis accurately be made preoperatively. Conservative surgery, chiefly vessel resection or ureteroplasty, is urged; nephrectomy is demanded with far-advanced renal destruction, infection or calculus disease. By specific example these cases serve to emphasize the great importance and necessity of the employment of modern urological methods in the diagnosis and treatment of persistent urinary tract disease in young patients.

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THE ROLE OF THE URETER IN RENAL TUBERCULOSIS

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THIS paper is intended to present a hypothesis based on quite generally accepted facts.

In 1917, Noel Hallé presented the first acceptable classification of renal tuberculosis on a biological basis. His work has since been repeated and amply substantiated by numerous investigators. All authorities agreeing as regards the acute miliary form, Hallé recognized only *two* types of *chronic renal tuberculosis*, viz.: Chronic closed parenchymatous renal tuberculosis and chronic open pyelitic renal tuberculosis. Note the careful incorporation of the adjectives "closed" and "open." It is the latter variety (chronic open pyelitic renal tuberculosis) that I wish to identify as the one here classed as "clinically established." It is frequently termed "surgical" renal tuberculosis. It is recognized as a progressive destructive and incurable lesion, and surgery is its only hope, and then only when the opposite kidney is free from the disease; otherwise the treatment must be only expectant and medical, awaiting ultimate death from tuberculosis.

SOME OF THE FACTS INVOLVED

A tuberculous kidney definitely discharging tubercle bacilli represents a progressively destructive process and there are few if any authentic records of complete healing in such a case. Many physicians still resist the significance of this well-known clinical fact. They seem to reason that because not infrequently complete healing does occur in other organs, therefore renal tuberculosis should not constitute an exception. If we can make them understand and appreciate the underlying reasons for this difference, perhaps there will be less temporizing in cases of unilateral renal tuberculosis at a time when nephrectomy would probably result in a cure.

In presenting the hypothesis submitted in this paper, other questions inevitably creep in, particularly that concerning the genesis and early developmental phenomena of upper urinary tract tuberculosis. Indeed it would seem probable that the same factors which predispose to this affection and are concerned in its etiology, are likewise important factors in its resistance to recovery. Again there arises the following pertinent query: being of hematogenous origin, why is the disease bilateral in only 35 per cent of cases? Are bacilli in sufficient numbers carried by chance to only one kidney? Or do the organisms reach both kidneys and gain a foothold in one only?

As has been particularly emphasized by Frank Kidd, probably most of us intermittently carry a few organisms, sometimes tubercle bacilli, in our blood stream, so that a kidney handicapped by slight hindrance to drainage with an increase in intrapelvic pressure, ever so slight, is apt, by selectivity, to become infected, while its fellow on the opposite side escapes or soon recovers. A striking example is unilateral typhoid pyelonephritis. When the blood stream is swarming with B. typhosus, why should one kidney escape? In one instance of unilateral typhoid pyelonephritis, the writer was enabled to indicate by means of ureteropyelography the plausible explanation and probable reason why one kidney became infected, and the other not: a handicap to drainage on the affected side by reason

of a moderate obstruction in its ureter, and hence its especial vulnerability.

By the same token it is logical to suppose that in a large percentage of cases of unilateral renal tuberculosis the bacilli reach both kidneys, but that in the vulnerable kidney the process is progressive while in the other the infection is overcome before it extends to the medulla. We can hardly escape the conclusion that when tubercle bacilli are present in the blood stream in sufficient numbers to infect a kidney, probably they are carried to the opposite kidney as well as to other organs; but that there exists a special reason or factor, which is often loosely referred to as local lowered resistance, that enables infecting organisms to gain a foothold in that particular kidney. It seems probable therefore in many instances that the determining factor in the genesis of unilateral renal tuberculosis is some degree of mechanical obstruction to the outflow of urine, difficult or impossible to demonstrate clinically.

There is much experimental evidence to show that by reason of the copious blood supply and the comparatively large caliber of the terminal renal vessels, tubercle bacilli introduced into the left ventricle or renal artery of healthy animals tend to pass through the terminal vessels and out into the renal vein, and then to lodge or be trapped in other organs, notably the liver, spleen, and bone marrow, without producing renal lesions. Supposedly (and to a certain extent experimentally proved), however, when there is an abnormality in the kidney affecting the blood vessels or glomeruli, a marked susceptibility to the disease is produced, or if in large masses, bacilli suspended in fat globules or adherent to debris, or agglutinated masses of bacilli, the organisms tend to lodge in the renal tissue as emboli.

When pus and tubercle bacilli of *renal* origin are found in the urine, renal tuberculosis may be said to be "clinically established." However, in practice, it must be determined by technical urological methods that these bacilli actually come from the kidney. As the years go by it is more and more the consensus of opinion of the most careful observers that there is no such thing as "excretory bacilluria," and that urine containing tubercle bacilli direct from the kidney and uncontaminated from any other source means an ulcerative lesion. In other words, if tubercle bacilli pass through a kidney from the glomerulus to the pelvis, there is always a tuberculous lesion in that kidney.

FACTORS WHICH DELAY HEALING BY CICATRIZATION

In certain organs and tissues of the body other than the kidney, recovery or complete healing by cicatrization is a not uncommon occurrence. Why the difference in the case of the kidney? The answer appears to be obvious if we may assume that recovery from a pyogenic infection of any organ is difficult or impossible if drainage is hindered. In this connection we might take note of the term "obstructive uropathy," coined by Young. It is a very expressive and significant designation of a fundamental etiologic and prognostic factor. Unless so mild that resolution occurs, practically all well-established bacterial infections anywhere in the body undergo recovery as the result of drainage either spontaneously by Nature's process, or through the intervention of the surgeon by means of incision, excision, or the introduction of hollow tubes through natural channels which are temporarily or permanently inadequate. Recovery does not take place without drainage, and with drainage other agencies usually are essentially superfluous and insignificant. Likewise, even an almost imperceptible hindrance to drainage, with an increase of only a few millimeters of pressure in a tubular or semitubular organ, is perhaps the factor that renders the organ more vulnerable to both pyogenic and tuberculous infection, as well as hindering its recovery when infected.

Renal tuberculosis is always secondary to a tuberculous focus elsewhere in the body and the infecting organisms almost invariably reach the kidney by way of the renal artery. The lesion originates in the parenchyma. The first gross evidence is observed usually in a pyramid near its base. So far as may be inferred from animal experimentation (and occasionally at autopsy) microscopic tubercles in the cortex may antedate this gross lesion in the pyramid by a considerable period of time. Until there is an ulcerative lesion, that is to say until the tuberculous process has extended to a calyx or to the collecting tubules (the "open" pyelitic lesion of Hallé and the "ulcerative" lesion of Medlar), tubercle bacilli are not present in the urine from that kidney, nor does the urine contain pus cells at this stage unless these pus cells arise from a pre-existing or concomitant non-tuberculous infection. Finding of acid-fast bacilli without pus cells is not evidence of an open pyelitic tuberculosis. At this stage perhaps the lesion may be so small that a prolonged arrest sometimes occurs with a remission of symptoms. In the prepyuric stage there are rarely any pronounced subjective symptoms or objective findings referable to the urinary tract. There may be a slight albuminuria or afternoon rise in temperature, seldom over 99.3° unless due to a tuberculous lesion elsewhere or other concomitant cause; rarely anything pronounced in the way of urinary symptoms until pus cells appear in the urine. Even a slight soreness over the kidney is uncommon in the prepyuric stage. Tenderness and pain over or in the tuberculous kidney means that the tuberculous process is not recent. Indeed, renal tuberculosis as we see it clinically has usually existed from one to three years before it has been definitely diagnosed. Excluding other etiologic factors, frank hematuria of renal origin means that the disease has already invaded, or by reason of a ruptured vessel has suddenly broken into, the renal pelvis.

When a tuberculous process once reaches a calyx, in other words when it has become an "open" pyelitic lesion, a new clinical picture, as well as characteristic pathologic changes, soon begins, due to a descending infection involving the ureter and bladder. Tubercle bacilli are now being excreted, whereas in the prepyuric stage these bacilli were not being released, at least they could not be discovered. The new clinical manifestations, essentially urinary, are frequency and urgency of greater or lesser degree, and pyuria, and these are the symptoms for which the patient usually first consults the physician. The condition is all too frequently diagnosed at this time as just "cystitis" without any adequate attempt to identify the infecting organism and to determine the primary focus in the urinary tract.

At one or more situations in its course, most often at its distal end, the ureter almost inevitably becomes involved, and eventually the site of definite pathologic changes, viz.: tubercles, infiltration, ulceration, thickening, fibrosis, and rigidity, which lead to constriction, of some degree at least, of its lumen at one or more sites.

Israel endorses Casper's statement that "in renal tuberculosis of long standing the ureters invariably become diseased." Those few (particularly Israel) who have investigated the subject by examining ureters removed routinely by nephro-ureterectomy in a large series of cases of renal tuberculosis, find this to be true. The fact that one notes no thickening or other pathological condition of that portion of the ureter which can be palpated and seen at the time of the nephrectomy, or that he can pass a catheter readily through such a ureter, is no evidence that the ureter is not tuberculous. Besides the narrowing or stricture, the elasticity of the ureter to some extent is lost and the peristaltic function is probably interfered with. As a result of a constricted ureter and defective peristalsis, a varying degree of back-pressure in the kidney is evidently produced. It is scarcely

rational to expect the recovery of a kidney handicapped by such hindrance to drainage of its never-ceasing secretions. Ureteral dilatation is of little if any lasting therapeutic value, in contradistinction to the results obtained by dilating strictures produced by a non-tuberculous process. Indeed, while there may be temporary improvement from such dilatation, instrumentation of the tuberculous area, as is the case with tuberculous lesions elsewhere, tends only to result in later progressive changes.

The point is often made that with rest, diet, fixation, heliotherapy, and other therapeutic measures, tuberculosis heals through the process of sclerosis, calcification, etc. In this respect, renal tuberculosis differs from that in practically every organ (except the meninges) where tuberculosis is common, because there can be no rest for either kidney or ureter. This, plus inelasticity or narrowing of the ureter, or both, seems to be the answer to the certainty of the kidney's ultimate destruction if the patient survives long enough. For if it were otherwise possible for any case of renal tuberculosis to heal completely, the tuberculous ureter would constantly threaten any remaining chance for the kidney to recover, even if nature had successfully negotiated all other hurdles. Renal tuberculosis may perhaps be so inherently destructive and progressive that in most cases the kidney could not recover even with an optimum functioning of the ureter. However, the clinically observed good results of ureteral anastomosis to skin (ureterostomy) when necessitated for purely palliative purposes in advanced renal and intractible *vesical* tuberculosis subsequent to nephrectomy for tuberculosis on the opposite side, amply demonstrate the important final role of the tuberculous ureter in the progress of the disease. We have repeatedly observed amazing benefit and remissions of years follow this procedure.

By careful section of tuberculous kidneys there is abundant evidence of healing, healed areas being found side by side with tubercles and all the other pathologic changes incident to tuberculosis; but in spite of these attempts at healing, new areas become involved and the tuberculous process in the kidney is always progressive once the tubercle bacilli reach the calyx. On the other hand, there is some supporting evidence (Chute, Hallé, Medlar, Thomas, and others) and much reason to believe that small tuberculous lesions in the renal cortex not infrequently heal completely; but never completely when the lesion has reached a pyramid, calyx, or the collecting tubules.

REMISSIONS

In renal tuberculosis it is not uncommon for remissions to occur. Although these remissions are sometimes of considerable duration, in rare instances as long as five years, they can only mean a temporary quiescence of symptoms, and in the end are always succeeded by a progressive destruction of the kidney. A remission does not mean that the tuberculous process in the kidney is healed, but simply that the activity has been slowed down. The pyuria does not disappear unless the ureter has become so strictured as to block off the kidney from the bladder, although it may be temporarily diminished. In that case, the bladder lesions may heal because of the fact that the bladder is no longer fed from above with urine containing tubercle bacilli. That is to say, the tuberculous bladder usually heals when the source of fresh infectious material is headed off. In such a case of pathologic or autonephrectomy it is probable that we can never consider the kidney a healed one. This is evidenced by the fact that in this condition when nephrectomy is not done, there is a large incidence of metastatic tuberculosis, such as acute miliary tuberculosis, tuberculous meningitis, etc.

SUMMARY

1. Where a tuberculous lesion has advanced to the renal pelvis or collecting tubules, renal tuberculosis never com-

pletely heals because of the handicap of hindered drainage and back-pressure, due to tuberculous changes in the ureteral wall; that is to say that, aside from other possible reasons, a tuberculous kidney excreting tubercle bacilli cannot recover because an incurably strictured ureter interferes with normal drainage and peristalsis.

2. When complete healing does occur, the tuberculous process is limited to the cortex, i.e., it has never extended to a pyramid or collecting tubules.

3. Until the tuberculous process has extended to a calyx or the collecting tubules, pus and tubercle bacilli of renal origin will not be found in the urine, and therefore, up to this time, renal tuberculosis may not be said to be "clinically established."

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ADVANCED BLADDER AND URETHRAL TUBERCULOSIS

TREATMENT BY URETERAL TRANSPLANTATION FOLLOWING PRELIMINARY COLOSTOMY*

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EXTENSIVE tuberculosis of the bladder that has failed to subside or has progressed following nephrectomy for clinically unilateral renal tuberculosis has offered a most difficult problem in treatment. The seriousness of this state of affairs is increased tremendously in those cases developing tuberculous stricture of the urethra. Whereas some of these cases clear up under ideal conditions of sanatorium care, the vast majority of them run a fatal course. The tremendous frequency of urination always present acts in a vicious way to extend the lesion. Diversion of the urinary stream is the one method of putting the bladder at rest. Prior to the development of ureteroenteric anastomosis this end was accomplished by nephrostomy or ureterostomy. The latter was generally regarded as being the operation of choice.

Ureter transplantation as developed by Dr. Robert C. Coffey would appear to offer an ideal method for the management of these cases. Some have been so treated by Dr. Coffey, as well as by other surgeons. The cases reported have done well in most instances. However, infections at the site of anastomosis and in the kidney by ascent following ureter transplantation are inevitable and their results are not to be minimized, particularly in this group of patients who at best are poor operative risks and unlikely to tolerate well pyogenic infections. These considerations have led many surgeons to feel that the risks entailed in transplanting the sole remaining ureter into the bowel were too grave to justify the procedure, if safer methods could be employed. In these cases where it is so essential to avoid infections, this can be effectively accomplished by performing preliminary sigmoid colostomy of the so-called double barrel type. Following colostomy the lower loop of bowel can be rendered sufficiently aseptic as to eliminate the hazard of infection from the subsequent ureter transplant operation. The following case illustrates the successful use of this method in a situation that appeared so desperate as to preclude the hope of a satisfactory outcome from any procedure.

H. W., No. 211506, aged twenty-seven, white male, married. The patient had a nephrectomy for left renal tuberculosis in 1929 at the University Hospital. Following an uneventful convalescence he returned to his home in Ohio. He re-entered the University Hospital May 6, 1931. At that time he had urinary incontinence and suffered continuous bladder pain. He had lost over 50 pounds of weight during the preceding year. He was greatly dehydrated and emaciated. The lungs were normal. The prostate and seminal vesicles were extensively involved with tuberculosis and there was a far advanced bilateral epididymitis. There was a tuberculous stricture of the entire bulbous urethra which admitted only a #10 French Coude catheter. A large perineal sinus communicated with the bulbous urethra. The bladder capacity was less than 2 ounces. Although he tolerated a urethral catheter poorly its inconvenience was more desirable than the bladder spasm experienced without it. He was therefore kept on indwelling catheter drainage. Narcotics were freely administered. Urograms were made following the intravenous administration of uroselectan. The right kidney and ureter were well visualized (Fig. 1). There was a marked hydronephrosis and hydroureter. The pyelogram revealed no characteristic changes of tuberculosis. The total phenolsulphonephthalein output for a two-hour period

* From the Department of Surgery, University of Michigan.

was 60 per cent. The blood non-protein nitrogen was 34.8 mg. per 100 c.c.

On June 18, 1931 exploration of the abdomen was made through a muscle splitting incision in the left lower quadrant. The bladder was small and hard and its surrounding tissues indurated. The right ureter was identified. It was enlarged but did not seem to be indurated. A portion of the sigmoid colon was brought into the wound and a colostomy of the double-barrel type was made. The postoperative course was uneventful. The colostomy was opened on the fourth postoperative day with a cautery knife. Following this the upper loop was treated in the usual way with enemas twice daily. The lower loop was irrigated every four hours with soapsuds enemas from above and drained from below by means of a rectal tube. These enemas were followed by prolonged douches with warm $KMnO_4$ solution of 1:5000 dilution. Shortly following the colostomy operation a second periurethral abscess developed. Treatment of this delayed his second operation seven weeks.

On August 11, 1931 a suprapubic incision was made. The intestines were packed upward in the peritoneal cavity. The wall of the upper rectum was now picked up and a longitudinal seromuscular incision 2 inches in length was made. The muscle coats were stripped back laterally from this for a distance of half an inch. At the lowest point in this area the mucosa of the bowel was opened to later admit the severed end of the ureter. This was done without fear of contamination although the area was kept well packed off. The parietal peritoneum overlying the lower end of the ureter was now opened and the ureter picked up and cut between clamps. The lower end was ligated and dropped back and the peritoneum closed. A soft rubber catheter of the Robinson type, size 16 French was now introduced 4 inches into the open end of the ureter and held in place by a triple 0 catgut suture which was run through a point in the circumference of the lower end of the ureter and then tied to it. This catheter was put in to insure drainage during the immediate postoperative period, and came away on the tenth postoperative day. The end of the ureter with its projecting 8 inches of catheter was now introduced into the lumen of the rectum and the usual Coffey technique of uretero-enterostomy followed. At this point an assistant introduced a proctoscope into the rectum and brought the end of the catheter out the anus. The abdominal wound was closed without drainage.

Fig. 1. Urogram made following intravenous administration of skiodan, before ureteral transplantation.

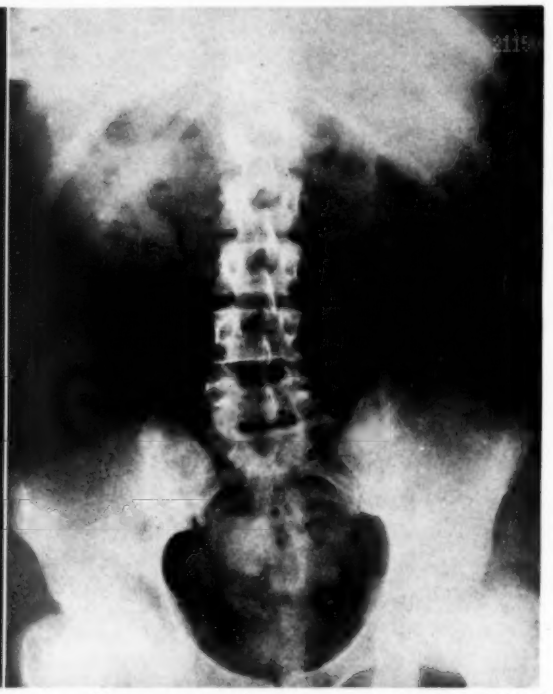

Fig. 2. Urogram made fourteen months following ureteral transplantation.

The postoperative convalescence was prolonged, due to necessary treatment of the genital lesions. The bladder was lavaged with boric solution daily as long as he remained in the hospital. A bilateral epididymectomy was performed during his convalescence. Upon his return home he was given full instructions regarding a proper regime to be followed. A check-up examination was made at the end of six months. At that time he had gained 40 pounds in weight, his appetite was good, and he was steadily gaining strength. The colostomy was functioning satisfactorily with two bowel movements daily. His urinary control was perfect. He passed his urine every two hours during the day and twice at night. Occasionally, if the rectum became unusually full of urine there was a slight overflow through the colostomy opening. This was never a troublesome factor. He had recently stopped lavaging his bladder because he felt it to be entirely healed. Examination revealed great improvement in the genital lesions. The perineal sinus while still present was almost completely healed. A catheter introduced into the bladder caused practically no irritation and upon lavage with boric solution the returned fluid was clear. Urine obtained from the rectum contained a moderate amount of mucus and a few pus cells and colon bacilli. Tubercle bacilli were not demonstrated by acid fast stains or by guinea pig inoculation. The patient was again sent home with orders to continue with his regime of treatment.

He was again seen in October 1932, fourteen months following the ureter transplant operation. At this time his total weight gain since operation was 63 pounds. The colostomy was functioning satisfactorily. He was holding his urine from two to four hours by day and was able to go through the entire night without emptying his rectum. He no longer had any reflux of urine through the colostomy opening. The genital lesions had all healed. The prostate and seminal vesicles had cleared up remarkably, but were not entirely free from evidence of infection. Urograms made following intravenous administration of skiodan showed a remarkable improvement in the kidney and ureter. (See Fig. 2.) There was at this time little evidence of hydroureter or hydronephrosis. As before there were no changes suggestive of tuberculous lesions in the kidney. The total PSP output for two hours was 60 per cent, the blood NPN was 38.6 mg. per 100 c.c. *X*-rays of the chest made at this time were negative for evidence of pulmonary involvement. The patient was sent home for another six months' period of rest treatment.

The outcome of this case appears hopeful for complete recovery. Should improvement continue, the question of closing the colostomy will inevitably arise. At present the patient is completely satisfied to leave matters as they are, and I am disposed to agree with him. However, should a reasonable desire to close it occur at some future date, there would seem to be no serious objection to doing so.

CONCLUSIONS

Progression of bladder lesions following nephrectomy for unilateral renal tuberculosis offers a serious obstacle to recovery. When complicated by tuberculous stricture of the urethra this condition is likely to terminate fatally.

Putting the bladder at complete rest by diversion of the urinary stream constitutes the soundest policy of treatment.

The most satisfactory methods for accomplishing this are ureterostomy or uretero-enterostomy. Of these, the latter is the more desirable procedure but carries the distinct hazard of infection in patients that are at best poor operative risks and tolerate infections poorly.

Colostomy as a preliminary measure before ureteral transplantation is proposed as a method for eliminating this hazard of infection.

The presentation of a case successfully treated by this method is made.

CARBUNCLE OF THE KIDNEY

(METASTATIC STAPHYLOCOCCUS ABSCESS OF THE KIDNEY)*

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THE term "carbuncle of the kidney" was first used by Israel[1] to describe a metastatic, hematogenous, localized infection of the kidney.

The first lesion he observed occurred simultaneously in a patient with a carbuncle on the back of the neck. He was struck by the similarity of the skin carbuncle and the kidney lesion. He therefore designated the lesion "carbuncle" of the kidney. His first observation was made in 1890 and the lesion was fully described by him in 1901.

If we are to judge by the literature that has accumulated in which infectious lesions of the kidney have been reported under this term, the condition is rare. However, it does occur frequently enough and presents sufficient clinical and pathological characteristics to merit recognition as a distinct clinical entity.

A review of the literature by four American authors reveals a rather large discrepancy concerning the number of cases reported. Neff[2] in May, 1930, collected from the literature 57 cases in determining the type of treatment employed by various men. Moore[3] in June, 1930, found 42 cases adequately described and referred to 22 in which there were insufficient data to be included in his series. He added 5 personal cases. Lipshutz[4] in December, 1930, in a similar review disclosed 58 cases. Brady[5] in March, 1932, in an extensive review of the subject recorded 88 cases, including his own.

Since his review, additional cases have been added by Mark[6] one; Fritz[7] one; MacMyn[8] 2; and Campbell[9] 2. The following case brings the total to 95.

A. B. White; school boy, seventeen years of age. Was admitted to the Medical Service September 11, 1930, complaining of a dull ache and tenderness in the left back and flank. He gave the following brief history.

On August 21, 1930, while practicing football, he was struck in the left side by the shoulder of an opponent and knocked to the ground. His side continued to hurt him but he resumed play; that evening he consulted a physician who believed he had fractured a rib (no *x*-ray); after his side was strapped, he experienced complete relief from pain for two weeks.

One week before he entered the hospital, or twenty-one days after his injury, his left side began to ache again. He noticed that if he remained quiet in bed, he suffered little more than an uncomfortable feeling in the side, but as soon as he was on his feet the aching returned and if he exercised very much, he experienced real pain in the back. It was always in the left costovertebral angle and flank and at no time was it referred to any other part of the back or abdomen until the day of admission to the hospital, when he experienced a severe colic referred to the left abdomen and groin. He did not experience any severe rigors but there were times when he felt chilly. He thought there were times during the week before admission when he had fever. He never observed any blood in his urine, but has noticed during the last week a nocturia (2–3) and slight burning on urination. The patient states that except for weakness, his general physical condition is good. He has lost no weight. Past surgical, medical and venereal history is negative.

Physical Examination: The patient was a tall, slim, muscular white boy, apparently about seventeen years of age, weighing 135 pounds. The physical examination was entirely negative except for tenderness elicited in the left costovertebral angle associated with moderate rigidity of the back muscles. The left side of the anterior abdominal wall, both in the upper and lower quadrants, as well as the left flank, were moderately tender. Deep palpation revealed definite rigidity in the same areas. Attempts at palpation of the left kidney met

* Read before the Columbus Academy of Medicine, October 24, 1932.

with marked rigidity, making it impossible to palpate it. Fist percussion over the left kidney resulted in a very definite painful response in the region of the left kidney. His extremities, genitalia and reflexes were normal.

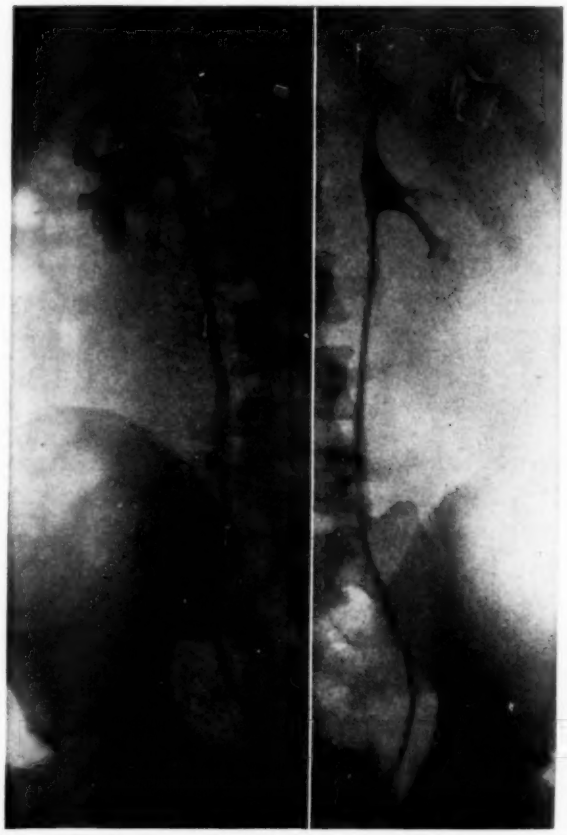

Fig. 1. Pyelogram of left kidney showing absence and deformity of calices.

Except for the loss of about 5 pounds in weight and an increased feeling of weakness, the patient feels better than when he entered the hospital twelve days previously.

His temperature on the afternoon of the first day in the hospital was 103°F., pulse 100.

There was a persistent afternoon rise in temperature, varying from 2° to 3° for sixteen days prior to operation. The urine upon admission was acid, sp. gr., 1.020, no albumin, sugar none, W.B.C. 1–3, R.B.C. a few.

Blood Count: September 12, 1930: Hemoglobin 96 per cent; R.B.C. 4,000,000; W.B.C. 15,100; polymorphoneuclears 71 per cent.

September 23, 1930: Hemoglobin 90 per cent; R.B.C. 4,800,000; W.B.C. 26,700; polymorphoneuclears 84 per cent.

Non-protein nitrogen: 37 mg. per 100 c.c. of blood.

Total *phenolsulphonphthalein* 65 per cent.

Blood sedimentation: very rapid.

Wassermann reaction: negative.

Fig. 2. Section of kidney through abscess.

Cystoscopic Examination: September 23, 1930. Normal bladder was observed; the ureters were both catheterized without obstruction being met. *Urine from the right ureter* was cloudy, alkaline, no albumin, W.B.C. none, R.B.C. few; P.S.P. appeared in seven minutes, output 16 per cent in fifteen minutes. No organisms were found on smear.

Urine from the left kidney was cloudy, alkaline, albumin positive, W.B.C. none, R.B.C. none; P.S.P. appeared in three minutes, output 10 per cent in fifteen minutes. No organisms found on smear.

Plain x-ray plate shows both kidneys to be in normal position, size and shape. There are no shadows suspicious of calculi.

Pyelogram of the right kidney was normal.

Pyelogram of the left kidney showed complete absence of the middle calyx. There was elongation and narrowing of the superior calyx. There was an exact reduplication of this pyelo-

gram following the administration of intravenous skiodan.

Diagnosis from pyelographic evidence: renal neoplasm. (Fig. 1.)

Operation: September 27, 1930. Left curved lumbar incision. Exposure of the fatty capsule of the kidney showed it to be dusky grey in color and quite edematous. The fat over the anterior surface and convex border of the lower two-thirds of the kidney was moderately adherent to the capsule. The capsule was torn in the process of removing the perirenal fat exposing the kidney which was studded with small abscesses surrounding a necrotic area about 1 cm. in diameter. The kidney was perceptably enlarged anteroposteriorly in this region. A semi-fluctuant mass could be palpated. We decided that the kidney was inflammatory and involved too much of it to be treated other than by nephrectomy.

The patient's postoperative course was uneventful. He was discharged from the hospital eighteen days following his operation in excellent condition.

Pathological Report. Specimen 6462. The kidney is moderately enlarged. The middle third is enlarged anteroposteriorly. Palpation of this enlarged area reveals a semi-fluctuant mass within the kidney parenchyma. The capsule overlying this region is thickened and torn; when removed there is revealed an irregular, dark necrotic looking area which overlies the deeper mass. Section of the kidney through the enlarged area reveals a necrotic, sloughing mass $3 \times 4 \times 4$ cm. (Fig. 2). There was a very small amount of free pus present. Two-thirds of the phlegmonous mass is located in the medulla and one-third in the cortex. It is fairly well demarcated from the surrounding kidney tissue which is compressed and gives the impression of encapsulation. The inflammatory process is progressing toward the cortex; the mass is composed of three necrotic areas surrounded by many smaller ones, which fuse indefinitely with the surrounding kidney tissue. The remainder of the kidney tissue is not remarkable.

The microscopic sections from this kidney show definite evidence of multiple abscess formation, there being a comparative large area of necrosis which is surrounded by a somewhat dense area of kidney tissue, which contains many small abscesses. The tubules show evidence of compression immediately surrounding the mass as they are flattened and surrounded by a large number of fibroblasts. The examination under high power of this region shows a considerable number of the tubules to be filled with polynuclear leucocytes with desquamation of the lining epithelium. The intertubular tissues contain an increased number of fibroblasts, increased vascularity with marked infiltration of polynuclear leucocytes.

The diagnosis is that of localized multiple abscesses with fusion into a large area of necrosis, or carbuncle of the kidney.

A smear from the lesion demonstrated staphylococcus. Culture proved it to be staphylococcus aureus.

ETIOLOGY

The condition is practically always closely associated with some infectious lesion which acts as a focus for blood stream invasion. The focus of infection may be present at the time the carbuncle develops or it may precede it by many weeks. Many times, infections of the skin precede the kidney lesion. Of the 95 cases reported, 70 per cent presented, at the time of the kidney lesion or before, some type of superficial infection, closely associated with the skin, such as furunculosis (35 cases), carbuncle (19 cases), infected fingers (5 cases), paronychia (4 cases), felon (one case), or a superficial abscess (one case). In 10 per cent the focus for distribution was thought to be respiratory (4 cases), tooth (one case), throat (2 cases), intestinal (one case), bone (one case) or glandular infection such as prostatitis (one case), orchitis (one case), parotitis (one case). In 20 per cent there was no mention made of any infectious lesion that could be held responsible for the origin of the blood stream invasion. It is possible that many of these patients had minor skin or other infections that antedated the kidney symptoms but had entirely forgotten them. Trauma in the renal region is held to be an etiological factor in connection with renal carbuncle, by Thompson,[10] Smirow,[11] Kohler,[12] and Dick.[13]

The fact that two-thirds of the cases reported occurred in the male might

possibly be taken as evidence that trauma plays some part in the etiology. We know that the male is more often exposed to varying degrees of extrinsic and intrinsic renal trauma than the female. However, trauma such as might arise from pregnancy, stone and urinary obstruction has been mentioned. These factors can be classed as etiological in that they produce a locus minoris resistentiae.

The classical experimental work of Brewer[14] demonstrated that a traumatized kidney is much more frequently the site of a localized infection than a sound one. In our case there was a history of definite trauma but we were unable to elicit a history or find present at the time of examination, a lesion that we thought could be held accountable as a possible source for blood stream invasion. Brady mentions 3 cases in which superficial skin infection was present at the time of trauma to the kidney in which the carbuncle developed.

The lesion was found to occur in the majority of the patients in the third and fourth decade. The two oldest individuals reported to have developed a carbuncle were fifty-five. (Zinn[15] and Reschke.[16]) Six cases have occurred within the first decade, three at ten years of age (Colmers,[17] Barth,[18] Beer[19]); one at eight and one-half months (Mark[6]) and two at eight weeks of age (Campbell[9]).

The disease is practically always caused by the Staphylococcus aureus. It is nearly always unilateral but Kretschmer[20] observed it as a bilateral lesion and found two other instances in the literature. The interval between the onset of the primary focus and the development of the renal lesion may be as long as seven months. The usual interim is one month.

PATHOLOGY

The development of a renal carbuncle presupposes a focus of infection, usually staphylococcic, with invasion of the blood stream and localized deposition of the organisms in the kidney parenchyma. The gross pathological feature resulting from such kidney invasion is a localized necrotic inflammatory mass in the substance of the kidney. The mass is made up of multiple foci of infection which is either definitely demarcated by a leucocytic or fibroblastic wall or which may fade without a limiting wall, into the surrounding healthy tissue. The lesion resulting is a phlegmon rather than a circumscribed abscess. The typical lesion early consists of a necrotic slough with free pus in small amounts scattered throughout the entire lesion. Abscess formation is a late finding and it is doubtful whether the diagnosis of carbuncle is justifiable when an abscess cavity is found. The carbuncle, usually located near one of the poles of the kidney, is often more cortical than medullary. The kidney is enlarged in the region of the lesion with moderate elevation of the cortex. Palpation of the lesion gives a sensation of a semi-fluctuating mass in the kidney substance. The progression of development is towards the capsule rather than toward the pelvis of the kidney. It may therefore perforate the capsule in one or more places with the development of a localized perinephritis or a perinephritic abscess.

The development of perinephritic abscess is many times secondary to renal suppuration. The etiology and mode of infection of both are very similar. The symptoms of the two processes are often indistinguishable and early differential diagnosis may be impossible. Richardson[21] in a review of 108 cases of perinephritic abscess, reported to be due to metastatic infection, found 24 of them due to extension of cortical suppuration. It is not expedient to investigate the kidney in every perinephritic abscess; if it were, primary suppuration of the kidney cortex would be more often reported as reasonable for perirenal infection. For this same reason, carbuncle of the kidney, we believe to be more frequent than reported. If the carbuncle is sufficiently walled off and there is adequate perforation

of the cortex for the slough to drain through, complete recovery follows drainage of the perinephritic suppuration; if it does not, further surgical investigation may reveal the carbuncle. It is interesting to note that in these cases of perinephritis, furunculosis preceded the abscess formation 55 times; paronychia or septic fingers, 21 times; carbuncles, 10 times; local abscesses 11 times; septic wounds and tonsilitis each 3 times. The organisms are carried by the blood stream to the kidney as a bacterial or infectious embolus. Mathe[22] believes that the organisms lodge in an end-artery, in a glomerulus in the cortex near the capsule and as the process continues a circumscribed infection of the adjacent renal cortex takes place, with fibrinous exudation, multiple foci of necrosis arise and the tissue adjacent to each necrotic plug becomes gangrenous. Barth believes that the bacteria lodge in the lymphatics of the renal parenchyma as he was never able to find them in the vessels of the glomeruli, but could find them in the connective tissue of the kidney. While the initial localization is in doubt, pathological studies suggest that the lesion is primarily one of multiple focal infection of the interstitial tissue of the kidney.

SYMPTOMS

The onset suggests an acute infectious disease with chills, fever and sweating, or it may be more gradual in its onset, suggesting a chronic infectious process, in which the first symptoms are headache, malaise, fever, anorexia and general weakness. These symptoms suggest a typhoid state. The acute stormy onset may suggest an influenza, pneumonia or other acute infections. Whatever the prodromal symptoms, the disease is not characterized by an early local, constitutional or urinary manifestations that suggest its presence. Either type of onset may precede the development of local symptoms, many days or weeks. However, the more severe the onset, the more likely the local loin symptoms will develop early. The fever is usually intermittent with a range of 101–103°F. The patients usually have an initial chill which may be repeated at irregular intervals or this phenomenon may be entirely absent. Our patient had no distinct chill although he complained of feeling chilly at times.

The local symptoms are those of pain, rigidity and tenderness in the costovertebral angle and flank. The pain is usually described as a dull ache or "gnawing sensation" in the back, with occasional attacks of sharp cramp-like pain that may be referred into the abdomen or groin. Our patient had nothing but backache when lying quietly in bed, but when on his feet this increased to a severe ache or pain. He did not have any sharp pain that was referred into the abdomen until the day before he entered the hospital. We believe this was due to the passage of a blood clot. Tenderness of the kidney is elicited on deep palpation early. Later, with the development of a subcapsular inflammation rigidity of the back and abdominal muscles may make palpation of the kidney impossible.

The development of a perinephritic abscess increases all local and constitutional symptoms with the added manifestation of a filled or bulging flank. A high leucocytosis is a constant finding.

The urinary findings are conspicuous because of their mildness or absence. Many of these patients have normal urine. This is explained by the fact that the lesion is in the parenchyma and extends towards the capsule of the kidney. The presence of blood and pus in large amounts may be encountered if drainage should take place into the pelvis. The nearer the lesion is to the cortex, the less likely there will be any urinary changes. Our patient had microscopic blood upon entering the hospital and ten days later a small amount of microscopic blood was found. The carbuncle in this patient was producing a definite pelvic distortion which accounted for the hemorrhage.

DIAGNOSIS

The diagnosis rests upon a carefully taken history and physical examination with particular care as to antecedent infections such as a boil, carbuncle or felon. These lesions should suggest the possibility of metastases of organisms to other viscera. A patient with a history of such a lesion who is either acutely or chronically ill with a septic temperature, high leucocytosis, flank pain, tender kidney, with few or no urinary symptoms establishes the possibility of a renal carbuncle.

The cystoscopic examination, as well as the urinalysis, may be negative. However, there is usually a decrease in the dye output on the affected side. Staphylococci may be cultured from the urine. Seldom are more than a few pus cells recorded in the catheterized urine.

The differentiation of carbuncle of the kidney from other focal infections of the organ such as solitary abscess, multiple abscesses or septic infarct may be impossible. Kretschmer believes that the smaller abscesses do not give rise to the same degree of resistance and fixation of the kidney on palpation as does the carbuncle. The lesion may be mistaken for appendicitis, cholecystitis or other intraperitoneal lesion if the peritoneum overlying the kidney is involved in the perinephritis. The differentiation of influenza, typhoid, subphrenic abscess, pleurisy, pneumonia or empyema from carbuncles of the kidney may be a difficult procedure, unless there are sufficient physical symptoms to suggest kidney pathology. The pyelogram may reveal a filling defect similar to a tumor of the kidney with absence, elongation or distortion of the calices, but it is rare for the lesion to produce pelvis distortion.

Brady in his review found that in only 14 cases had a pyelogram been made. In 11 of these there was evidence of abnormality. In our case (Fig. 1) there was an absence of the middle calyx, elongation and narrowing of the superior one, not unlike that seen in renal neoplasm. Similar pyelographic evidence has been recorded by Brady, Mathe and Moore. Evidence such as this has led to the diagnosis of tumor of the kidney, notwithstanding other clinical evidence to the contrary. The pyelographic evidence determined a diagnosis of renal tumor in our case.

TREATMENT

Judging from data in case reports and the opinion of the majority of authors, treatment of carbuncle of the kidney is primary nephrectomy. Forty-two cases were so treated in this group with 3 deaths, a mortality of 7.1 per cent. Incision of the carbuncle does not seem to give adequate drainage to insure a cure, and if it does, the disease may continue to develop by circumscribed extension, although drainage of the original lesion is adequate. In the 8 cases in which it was tried in this series, a secondary nephrectomy became necessary with one death. When there is a perinephritic abscess present, incision and drainage is indicated. Fifteen of these patients were so treated, with 3 deaths.

If a carbuncle is found, excision, enucleation or nephrectomy should be carried out. Excision of the carbuncle as a secondary procedure has been reported twice without a death; enucleation of the carbuncle followed by a nephrectomy is reported twice with one death.

Certainly the choice of surgical procedure is primary nephrectomy, providing the general condition of the patient will permit it. The possibility of excision of the lesion depends upon the location and amount of kidney tissue involved. Successful enucleation of the carbuncle depends upon the degree of encapsulation.

COMMENT

When you consider the frequency of the superficial and deep staphylococcal infections, you are immediately struck by the small number of renal and perirenal secondary infections reported. Certainly the kidney has to make some disposition of these organisms very frequently in every

individual. It stands to reason then that there are many grades of virulency of the organism, as well as immunity of the individual. These two factors reciprocally hold the gross lesions to a minimum. Obviously some carbuncles are never diagnosed and recover spontaneously, and many recover following drainage of the perinephritic suppuration. We only see the large suppurating lesions which produce definite and continuous signs and symptoms. We are disposed to believe that there was subcapsular injury of the kidney tissue with a deposition of staphylococci which became enmeshed within the interstices of the injured tissue in this case. However, careful examination gave no evidence of perirenal or intrarenal hemorrhage either recent or remote. It is possible that these organisms might have been successfully disposed of by an uninjured kidney.

CONCLUSIONS

Carbuncle of the kidney is accepted as a distinct pathological and clinical entity; it is a localized phlegmon of the kidney. There are no clinical symptoms or physical findings which differentiate it from other hematogenous infections of the kidney. The diagnosis is difficult but is suggested by the presence or antecedence of a superficial staphylococcal infection, usually of the skin. Trauma has seldom been recorded as playing an important role in its production.

Herein is reported a case in which there was no antecedent infection; there was a very definite history of trauma, a microscopic hematuria, a pyelogram which showed pelvic distortion and an incorrect diagnosis of renal neoplasm. Treatment consisted of nephrectomy with complete recovery.

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URACHAL CYSTS AND FISTULAE*

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CLEVELAND, O.

THE urachus is a rather vague entity in the minds of most surgeons like other vestigial structures which perform their major function and undergo their development largely in the primeval ontogeny of the human embryo. Its anatomical variations are promptly forgotten or dismissed from mind because its surgical importance is considered slight. Accordingly, fistulae, or infected cysts in abdominal wounds resulting from surgical injury to the urachus are uncommon and reports of their occurrence are exceedingly rare in the literature. The urachus is undoubtedly torn or cut innumerable times when midline abdominal incisions are made. Surgeons have a tendency completely to disregard the urachus which is rudimentary and obliterated normally at least in its major portion as it passes external to the peritoneum on its way to the umbilicus. The recent occurrence, however, of injury to the urachus giving rise to prolonged infection in two midline incisions has called our attention to this surgical complication.

It seems worth while to consider for a moment the development of the urachus and its failures of occlusion to understand the lesions which may occur as a result of injury. The allantois, or forerunner of the urachus, as well as the urinary bladder, appears very early in the development of the embryo. Ahlfeld, Ledderhose, and Vaughan, among others, early described the embryology and pathology of the urachus. The allantois has already appeared in an embryo of under 1 mm. as a projection of the yolk sac into the body stalk. As further development takes place (Cullen) the yolk sac has been incorporated in the embryo forming, at its lower portion, the hind gut. The allantois presents itself as opening into the hind gut (Fig. 1). In a later embryo the allantois is still in contact with the cloaca but curves upward and outward. Eventually it is separated from the bowel at its lower portion being converted into the bladder, and its upper portion is much narrowed, passing out into the umbilical cord and lying between the umbilical arteries. Its patency varies much in the embryo, in the umbilical cord, but it becomes impervious finally at the point of the emergence from the abdomen, i.e. at the umbilicus. There may be dilatations both in the intra-abdominal and extra-abdominal portions of the allantois. This has been demonstrated in an embryo 5.2 cm. As the embryo becomes larger, the urachus is found to be small and almost solid normally. It is seen therefore, that the allantois is one of the first definite structures to be delineated and that it is derived from the yolk sac. It is carried downward, coming from the cloaca, and the bladder develops from its lowest portion. The upper part of the allantois continues its course from the tip of the bladder to the umbilicus and perforates it, passing into the cord. The intra-abdominal portion is now called the urachus. The allantois and urachus become solid cords in part, but show, in various cross-sections, spindle-like dilatations (Fig. 2). As Cullen states, these dilatations are characteristic of the urachus and allantois and undoubtedly persist in many individuals, accounting for small cysts not infrequently noted at operative incisions made in the midline. Total patency, of course, accounts for the cases of congenital urinary fistulae at

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the umbilicus. (Fig. 2.) The urachus is stretched out and produces narrowing of the upper part of the bladder as it descends below the umbilicus. The bladder later descends farther after the birth of the fetus and the urachus is taken along below the umbilicus. The umbilical arteries are pulled along with it and the fibrous tissue of the umbilicus is drawn down with the tip of the urachus, hence the urachus is ordinarily described to be 5–6 cm. in length passing upward from the anterior bladder wall at its apex, usually ending at this point 10–12 cm. below the umbilicus and connected with it by its single cord of connective tissue. The urachus ordinarily consists of several layers with a circular epithelialized canal (in part of its length) and an outer connective tissue covering. Its canal communicates, as stated by Begg, in about ⅓ of cases with the interior of the bladder. Generally, the urachus does not communicate with the bladder but fails to perforate the vesical mucosa stopping just outside its external margin. In such cases where communication does exist and the urachus is opened, effusion of minute amounts of urine in the preperitoneal space may give rise to infection. These occur as "septic urachal cysts." It is not our purpose to discuss congenital urachal fistulae where there is actual and continuous urinary leakage, often in large amounts from the urachus, but merely to point out the fact that in a certain percentage of cases, injury to the urachus even if its communication with the bladder is slight, may give rise to annoying postoperative complications. Non-descent of the bladder in embryonic life may result in full blown vesico-umbilical fistulae, and as might be expected, fistulae into abdominal wounds lower down may result from injury of the patent terminal end of the normal urachus as well. The urachus lies in close proximity to the external surface of the peritoneum and is surrounded by preperitoneal fascia and is rather intimately adherent to the external surface of the peritoneum. Commonly two conditions

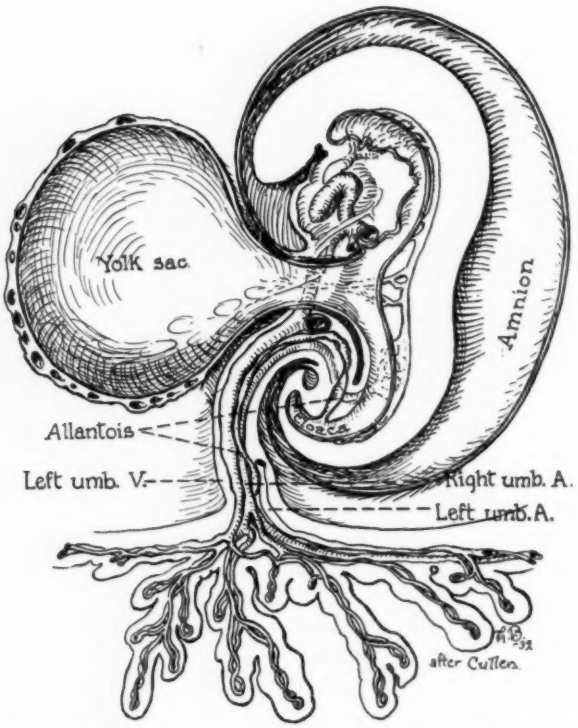

Fig. 1. Development of allantois in embryo of 2.5 mm.

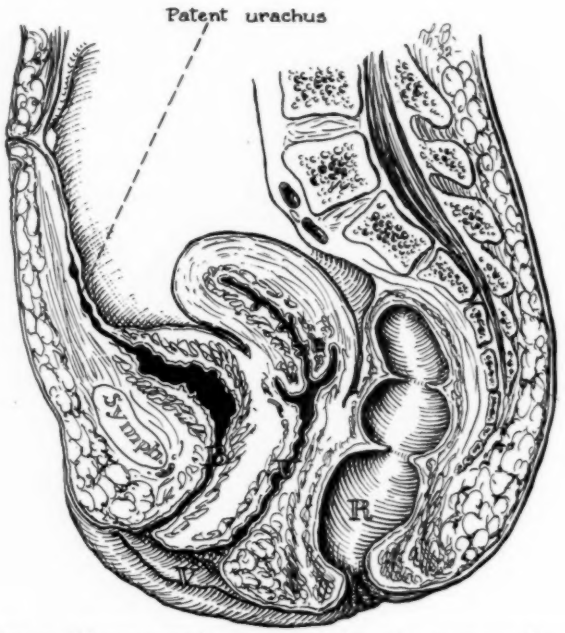

Fig. 2. Diagrammatic representation of patent urachus with cystic dilatation.

are found depending upon the manner and amount of obliteration of the urachus. (1) It may be a condition of complete persistence, in other words, the congenital urachal fistula which really is undescended or incompletely descended bladder with urinary leakage at the umbilicus through its tip the urachus (Fig. 2). (2) Escape of urine may occur through the terminal end of the urachal canal which has not become obliterated. This may be very small but may lead to an abscess in the prevesical space or in the abdominal wall below the umbilicus. The occurrence of infection in two wounds, recently due to contamination by urinary leakage through the injured urachus has called our attention to this lesion.

Case 1. The patient was a white woman aged fifty-one, admitted for a hernia of a low midline scar. She had had a pelvic operation in 1923. The wound had drained for nine months and finally healed. At the time of admission, the patient's abdomen was thick-walled, and the abdominal wound showed a large irregular hernial defect. At operation it was found that the sac contained no adhesions of omentum or bowel. The whole scar was excised adjoining the defect. The urachus was found to be involved in the scar, was divided and ligated and herniorrhaphy carefully performed. The patient was discharged from the hospital with the wound healed but after a month she developed a small hard mass in the upper portion of an otherwise normal well-healed midline scar. This was incised and 150 c.c. of pus evacuated from a small buttonhole in the fascial scar. This was drained and the wound healed. The patient has since been symptom-free, and the wound has shown no further signs of infection. The original hernia was presumably due to infection through the urachus. Persistent draining of the wound for a period of nine months showing no intraperitoneal adhesions, suggested especially since a rather large urachus containing a small lumen was opened in freeing the scar, that contamination had been of urinary origin through a slightly or intermittently patent urachus.

Case 11. This patient was a thirty-two year old colored nullipara entering the hospital complaining of bloody vaginal discharge, chills and fever, loss of weight and abdominal pain for a year. There had been a previous laparotomy for pelvic inflammatory disease four

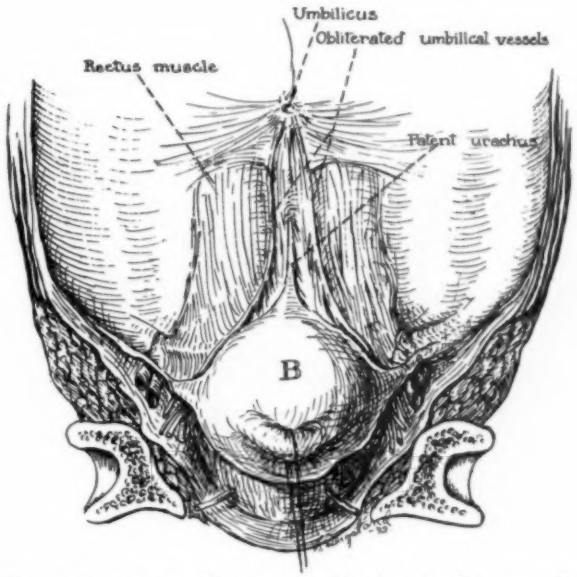

Fig. 3. Patent urachus apparently involved in scar in Case 11.

years before. The abdominal wound drained for six months and had never entirely healed. There had been persistently a slight drainage of the seropurulent fluid from a small point in the wound. Physical examination was negative except for fullness and tenderness on palpation in both lower abdominal quadrants. On pelvic examination the cervix was flat, infiltrated, rubbery in consistency and in appearance suggesting a granulomatous lesion rather than a true neoplasm. There was bleeding from the cervix. Both fornices were infiltrated and seemed almost immovable. A clinical diagnosis of tuberculosis of the cervix was suggested, which was sustained by biopsy. The cul-de-sac was comparatively clear. The patient's condition seemed progressively unfavorable and exploratory laparotomy was decided upon. At operation, injection of the sinus tract in the midline incision with dye demonstrated that the slight drainage from the abdominal incision was due to a urachal fistula which ran directly down into the apex of the bladder and communicated with it. Its lumen was exceedingly small and moderate pressure was necessary to force the dye through. It was obvious, however, that intermittent urinary drainage through the urachus had caused the incision to remain infected (Fig. 3).

CONCLUSION

In these two cases, it seems likely that injury to the urachus was the condition responsible for the midline wound infection which persisted apparently without cause over a long period of time, and the generalization may be made that very slight urinary leakage may be responsible for similar lesions and for hernias occurring in obscurely infected wounds as well. It seems worth while therefore in making gynecological incisions, if the urachus cannot be pushed to one side or the other and is not clearly obliterated especially at its lower portion toward the bladder, that it should be picked up and if there is the slightest question of its possible patency, should be ligated firmly.

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PANHYSTERECTOMY

CLINICAL ANALYSIS OF 150 CASES FROM THE VAGINAL AND THE ABDOMINAL APPROACH*

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PANHYSTERECTOMY, which can be done either through the vagina or the abdomen, is one of the few major operations which offers to the surgeon two different avenues of approach. As the end-result of either procedure, extirpation of the entire uterus, is the same, we have analyzed a series of 150 panhysterectomies in an attempt to determine which is the better method of approach.

Because of the frequent misuse and misunderstanding of the terminology dealing with operations on the uterus, a definition of the terms used is offered at the onset. Abdominal panhysterectomy designates extirpation of the entire uterus, including the cervix, by the abdominal approach; vaginal panhysterectomy refers to the removal of the uterus and cervix through the vagina. The adnexa are separate considerations, and if any part, or all, of the adnexa is excised at the time of the panhysterectomy, the adnexal procedure should be specifically designated.

Today hysterectomy is one of the most common, because it is comparatively safe, and perhaps the most abused, because it is technically easy, of the major abdominal operations. However, it was only forty years ago that hysterectomy was looked upon with "fear and trembling," and Goodell, as late as 1891, taught that only after the failure of all other procedures should hysterectomy be resorted to for uterine fibroids. Should we of today not listen to the voice of the past and be less quick to unsex a woman, be slower in performing a hysterectomy unless definite indications are present, and give more consideration to the physiologic function of the uterus rather than to anatomic extirpation?

The annals of hysterectomy in this country include many notable names, and three of these at least should be mentioned: Stimson, who taught preliminary ligation of the uterine blood supply as the method of controlling hemorrhage; Baer, who demonstrated the present technique of closure of the cervical stump, and H. A. Kelly, who perfected uterine rotation and bisection in order to facilitate operation on large tumors.

The indications for hysterectomy are many and varied, and change in their intrinsic aspects according to the times and the individual operator. In general, however, the type of operation should be that best fitted to assure the future welfare of the patient. There are many uterine conditions in which the abdominal approach for panhysterectomy is indicated, as in exceedingly large fibroids, adnexal operations with exploration of the abdomen, etc., whereas vaginal panhysterectomy should be the procedure of choice in complete procidentia of the uterus, especially in elderly women. There is a vast, indeterminate field between these two definite indications, in which are included many conditions where panhysterectomy can be performed either vaginally or abdominally. The purpose of this paper is to endeavor to weigh the merits of both methods of approach.

From about 5400 abdominal hysterectomies collected from the literature, Fullerton and Faulkner estimated that hysterectomy in the best hands should have a mortality not over 2.5 to 3 per cent in selected cases, but Miller has ably

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shown that with the average surgeon the mortality is never under 5 or 6 per cent, and may run as high as 8 to 10 per cent. However, the mortality alone does not tell the whole story; morbidity, immediate and late, and postoperative complications should be carefully considered, but unfortunately are rarely discussed.

Vaginal panhysterectomy attracted much attention thirty years ago. It was performed by leaving heavy clamps on the broad ligaments for forty-eight hours, since ligation was considered too uncertain, and no effort was made to close the peritoneal cavity. Gradually this operation was discarded as the procedure in abdominal panhysterectomy was perfected. With the development of the new vaginal technique (Mayo), which unites the broad ligaments in the midline and furnishes excellent bladder support, vaginal panhysterectomy has grown to have a much wider field of usefulness than the average surgeon is willing to admit. In trained hands, such as Babcock's, fibroids as large as six month pregnancies, tubes, ovaries, pedunculated growths, ovarian cysts, appendices, and ectopic pregnancies have all been removed through the vaginal route. On the other hand, Babcock points out that the vaginal approach imposes operative difficulties on the surgeon trained only in the abdominal technique, and he advises that the operation need not be a blind one because of the limited operative field, but that the operator must be content to see and work on only a small amount of tissue at one given time.

Horsley favors the vaginal operation for panhysterectomy in cancer of the body of the uterus, and in uterine infection, since the abdominal approach presents the possibility of spilling cancer cells or bacteria along the route from the vagina to the abdominal wall. He also emphasizes that drawing upon the uterus squeezes out cancer cells or bacteria into the lymphatics. On the other hand, vaginal panhysterectomy in these two conditions offers an intact vaginal mucosa, if any cancer cells or bacteria are spilled, and downward traction on the uterus facilitates lymphatic drainage in this direction; consequently the spread of cancer cells or infection into the deeper lymph spaces is prevented.

That vaginal panhysterectomy has a limited surgical scope with the average operator is generally accepted, but this fact does not detract from the operation whose route for many pelvic troubles is the natural and logical one. Inversion of the uterus, uterine prolapse, fibroids of moderate size associated with procidentia, are all best treated by vaginal panhysterectomy, while the same route is the procedure of choice in obese, elderly women, often past the menopause, in whom postoperative hernias and immediate postoperative complications must be considered as dangerous possibilities. If, in such poor surgical risks vaginal panhysterectomy is the procedure of choice, we feel it should be all the greater choice in a patient with a similar condition who is younger, thinner, and generally a better surgical risk. In the great majority of cases vaginal panhysterectomy must be followed by a perineorrhaphy. The type of the perineorrhaphy is governed by the degree and extent of the perineal injury.

A consecutive series of panhysterectomies, 73 vaginal and 77 abdominal panhysterectomies, are analyzed from a clinical point of view, and only external constitutional factors, i.e., diabetes, cardiac disease, etc., influenced the omission of any case.

AGE

Abdominal Panhysterectomy	
Oldest	64
Average	44.4
Youngest	23
Vaginal Panhysterectomy	
Oldest	75
Average	50.2
Youngest	30

The average age of the patients was six years greater in the vaginal group; this factor should definitely influence both the morbidity and mortality figures,

since the operative risk is increased with the addition of years.

It was desired to ascertain more of the physical characteristics of the patients, that is, obesity, thinness, good or poor surgical risk, etc., but due to the lack of such data in the histories, these findings are omitted.

INDICATIONS FOR OPERATION

Abdominal Panhysterectomy

Fibroid	28
Cervicitis	19
Hyperplasia	14
Carcinoma of fundus	12
Endometrioma	6
Carcinoma of cervix	6
Ovarian cyst	5
Adenomyoma	4
Cervical polyp	3
Carcinoma of ovary	3
Fibrosis	2
Sarcoma of uterus	1
Chorioepithelioma	1
Pregnancy	1

Vaginal Panhysterectomy

Cervicitis	44
Uterine prolapse	35
Rectocele	19
Fibroids	16
Cystocele	15
Hyperplasia	13
Carcinoma of cervix	5
Endometrioma	4
Pregnancy	2
Fibrosis uteri	2
Cervical polyp	2
Carcinoma of fundus	1
Chorioepithelioma	1
Ovarian cyst	1
Acute endometritis	1
Vaginitis	1

Fibroids were the outstanding indication for abdominal panhysterectomy, although malignancy was associated in 21.3 per cent. This is a rather high figure as compared with the 4.3 per cent as seen in the vaginal group, and, in consequence, the mortality and morbidity in the abdominal series should be the greater. Cervicitis was the most frequent indication for vaginal panhysterectomy, although perineal relaxation in the form of uterine prolapse, rectocele and cystocele was present in 42.6 per cent. In view of the infrequency of chorioepithelioma it is striking to find this condition associated in each of the groups.

The type of anesthesia was about equally divided between the two series; spinal was used 6 times, and local 3 in the vaginal operations, while spinal was employed in 5 cases in the abdominal group. In all other cases general anesthesia was administered.

OPERATIVE PROCEDURES

Abdominal Panhysterectomy

Panhysterectomy	77
Bilateral salpingo-oophorectomy	44
Appendectomy	22
Unilateral salpingo-oophorectomy	5
Dilatation and curettage	3
Perineorrhaphy	2

Vaginal Panhysterectomy

Panhysterectomy	73
Perineorrhaphy	48
Anterior colporrhaphy	5
Bilateral salpingo-oophorectomy	1
Excision of vaginal fistula	1

Fifteen operators performed the abdominal panhysterectomies, with one surgeon doing 50 per cent of the operations; in the vaginal group only nine different doctors operated. The same surgeon who did the greatest number of abdominal operations also performed 63 per cent of the vaginal series.

The average operating time was twelve minutes shorter in the vaginal group, 43.4 minutes to 55.6 minutes, than in the abdominal series. About an equal number of patients, 46, were packed in each group.

POSTOPERATIVE TEMPERATURE

Abdominal Panhysterectomy

Average height	101.4°F.
Average	100
Average duration of fever in days	7.8
Average hospital stay in days	15.8

Vaginal Panhysterectomy

Average height	101.5°F.
Average	100.2
Average duration of fever in days	8.3
Average hospital stay in days	15.4

The striking observation concerning the temperature chart is the similarity between the two series, yet the abdominal panhysterectomy cases presented many more

complications, and three times the mortality, noted in the vaginal group.

COMPLICATIONS

Abdominal Panhysterectomy

Wound abscess	8
Cystitis	2
Diarrhea	2
Pyelitis	1
Perineorrhaphy broke down	1
Hemorrhage	1

Vaginal Panhysterectomy

Pelvic abscess	4
Cystitis	3
Hemorrhage	3
Vesicovaginal fistula	1

It was desired to include in the complications such treatment as gastric lavage, catheterization, saline infusion, etc., in order to portray a better picture of the immediate convalescence, but accurate information was not available from the histories. The high percentage, 10.4, of abdominal wound infection well substantiates Horsley's belief that bacteria are spilled en route from the vagina to the abdominal wall. Hemorrhage, as is the usual occurrence, was more frequent in the vaginal group.

MORTALITY AND CAUSE OF DEATH

Abdominal Panhysterectomy—6

Surgical shock	3
Intestinal obstruction	2
Acute dilatation of stomach	1

Vaginal Panhysterectomy—2

Peritonitis	1
Pneumonia	1

Six patients died in the abdominal group, a mortality of 7.9 per cent. Two of these, however, had a cancerous condition, and, as has been said, it is a far different thing to operate for cancer than for a benign tumor. In order to make the comparison of the mortality more uniform, the abdominal mortality, excluding carcinomatous conditions, could be reduced to 4 deaths, or 5.3 per cent, as against 2 deaths, or 2.7 per cent in the vaginal group.

SUMMARY

Vaginal panhysterectomy is by no means as popular as it once was, but the operation has many advantages: It can be performed under local, spinal or gas anesthesia, and the time required for the operation is, in general, shorter, even though a perineorrhaphy be included, than that consumed in abdominal panhysterectomy, since suturing the abdominal wall is eliminated. Uniting the broad ligaments in the midline offers excellent vesical support, and thus excludes any cystocele or possible recurrence of prolapse. Vaginal panhysterectomy avoids secondary complications such as abdominal hernias, parietal adhesions, and infections; there is no abdominal scar, and the convalescence and hospital stay are generally reported shortened, even though such was not the case in this series.

As already brought out, we feel that vaginal panhysterectomy offers an operative scope more extensive than the average surgeon is willing to admit, yet its field of operation is limited, and cases should not be selected in which this procedure will defeat its own purpose through the introduction of trauma and mechanical difficulties. Although easily borne by the patient, vaginal panhysterectomy may be much more trying on the surgeon than the abdominal panhysterectomy. The type of operation proposed should be that ideally best fitted to the patient, and not to the operator. The analysis of these figures is not a condemnation of abdominal panhysterectomy, an operation which is fully accepted and standardized, but an attempt to give full credit to the vaginal approach, an operation whose scope is not fully appreciated.

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HEMATEMESIS FROM PHYTOBEZOAR*

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SEVERE hematemesis as a symptom of phytobezoar has been rarely noted. Its occurrence in 2 cases personally examined by roentgen ray by the author is the chief reason for this report as it seems highly probable that through errors in diagnosis many patients with this condition are denied the opportunity for recovery which only operative treatment can give.

The occurrence of phytobezoar is considered to be relatively rare in the human patient and the possibility of such a tumor is quite generally ignored by medical textbooks, but it is probably more frequent than has been supposed since the natural course is to advise against an exploratory operation after the demonstration of a large gastric tumor. Indeed, palpation of the stomach during a laparotomy may fail to give evidence of such a mass (Hart[1]). The cases of which reports have been found do not therefore fairly represent the incidence and more instances will no doubt be recorded as attention is directed to the condition.

Hart,[1] Maes,[2] Balfour and Good[3] and Potter[4] have reviewed the literature up to a recent date and for the sake of brevity the major portion of their references has been omitted from the appended bibliography. In addition to the cases they have described and those they have reviewed, we find additional case reports by Tschassownikoff,[5] Adams,[6] Prasad,[7] Mambrini,[8] Droegemueller,[9] Camp,[10] Pollok,[11] Hamdi,[12] Cole[13] and Wyatt,[14] so that the known number in the literature rises to 50 cases, exclusive of an indeterminate number of "Harpanahalli bezoars" discussed by Nathan.[15] Three of these bezoars, those reported by Nifong and Blesh (V. David[16]) were discovered in the ileum.

An extended discussion of the etiology, symptoms, roentgen-ray findings and differential diagnosis will not be entered upon here. Briefly, bezoars are of two general types: the trichobezoars and the phytobezoars, made up of hair and of vegetable fibers, respectively. Mixed types may occur. In the human patient the former are found chiefly in neurotic or insane women. The latter may occur in any person but usually follow the use of persimmons in the diet, these being especially likely to lead to bezoar formation on account of their high content of gum and pectin.

Nathan[15] has described a bezoar peculiar to one part of India. Another type of mass may be found, formed by precipitation of shellac from its alcoholic solution when such has been used as a beverage. An instance has also been reported in which raw caoutchouc formed the matrix (Prasad[7]). The 8 phytobezoars reported by Hart form the largest single collection in the literature, although these did not all occur in his own practice.

The symptoms have been described as those of an acute and severe gastroenteritis (usually occurring shortly after eating persimmons), attacks of diarrhea alternating with constipation, or possibly only a sense of epigastric discomfort. Food relief is inconstant. Peptic ulcer was found in 2 cases reported by Hart and in 2 cases reported by Pollok and Cole. Balfour and Good call attention to the ease with which any gastric trauma may cause gastric ulceration.

Vomiting may not be a prominent symptom. Occult blood may be present in the stomach contents and stools. All or part of the mass may pass into the small intestine and produce symptoms of obstruction. In the typical instance the gen-

* From the Department of Roentgenology, University of Indiana School of Medicine.

eral nutrition remains good until the late stages.

The roentgen-ray findings are those of an intragastric mass which is usually not tender. There should, of course, be no pedicle demonstrable. Although it has been stated that displacement by manipulation is a diagnostic point, such was possible in only one of these two patients, and in that one at the first examination only.

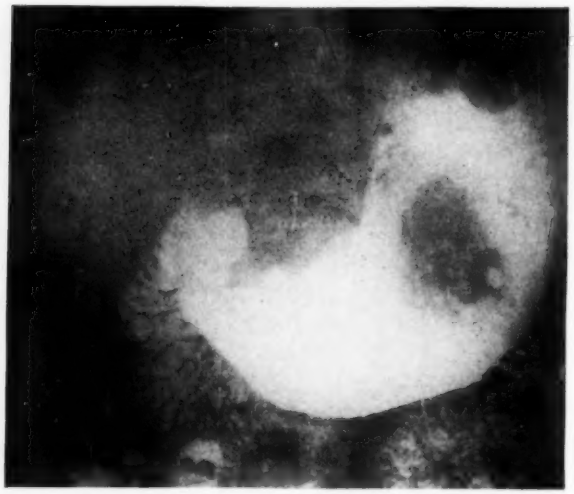

Fig. 1. Roentgen appearance in Case I.

CASE REPORTS

Case I. A man, aged fifty-three, was referred for roentgen study on account of tenderness and continuous severe burning pain in the left hypochondrium. Some relief had been afforded by liquids, but none by food or alkalies. There was considerable anorexia and weakness. Some vomiting had occurred, beginning one week previously and evidently containing blood. There was neither constipation nor diarrhea. A large intragastric tumor was present in the pars media (Fig. 1), with constant irregularity of the lesser curvature for about 3 inches proximal to the pylorus through which peristaltic waves were not traced. Six-hour motility was normal. As no pedicle could be visualized on the mass, a phytobezoar was suspected but since the patient denied eating persimmons there seemed to be no sound basis for such a diagnosis and a report of probable malignancy was made.

Within a few days the patient was admitted to the Indianapolis City Hospital as an emergency case on account of severe hematemesis. A laparotomy was performed by Dr. Karl Ruddell and a large food-ball was removed from the stomach. The gastric mucosa was edematous but no other lesion was found. Uneventful recovery occurred. By this time the patient recollected that about a week prior to our first examination a large quantity of persimmons had been eaten. He felt ill that night and from then to the time of operation he could take only a few bites of food at a time. The patient was seen again six years later. During the interim he had been symptom-free. Normal findings were obtained upon roentgen study of the stomach at this time.

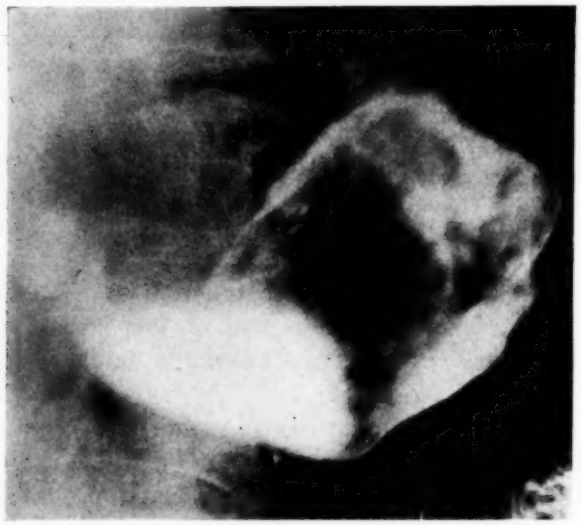

Fig. 2. Roentgenogram showing phytobezoar in Case II.

Case II. A man, aged fifty-three, entered the hospital on account of sudden, severe hematemesis. For several years he had had pain and tenderness in the epigastrium, nausea, vomiting at intervals and constipation. For three or four months prior to the hemorrhage there had been marked and increasing pain in the abdomen, differing in nature from his previous symptoms, almost constant and quite unrelated to food intake or bowel movements. Otherwise the history as obtained was negative. After the patient had recuperated from his loss of blood we made a roentgen study and found evidence of an intragastric tumor which had no demonstrable pedicle and which could be displaced upward into the fundus (Fig. 2). A niche defect was found on the lesser curvature. Direct questioning at this time brought information that for some years he had eaten

large quantities of persimmons in their season. A diagnosis of phytobezoar was therefore made, as well as of an ulcerative gastric lesion, probable cholecystitis (upon results of Graham dye test) and disease of the appendix. At a confirmatory examination made a few days later the tumor could not be displaced by palpation, probably because there was more pain on pressure and consequent abdominal rigidity. Two months later a laparotomy was performed by Dr. Ross Ottinger and three foodballs were removed from the stomach. A large ulcer was found to have perforated into the pancreas with the appearance of inoperable malignancy and as the patient was not in good condition no further surgical procedures were attempted. The roentgen findings of chronic cholecystitis and appendix disease were also confirmed at this time.

Twenty-seven months later the patient returned for further roentgen study at our request. During the interval he had neglected all medical treatment. At this time he had dull, heavy pain at the umbilical level, not severe but present during all his waking hours and not relieved by food, alkalies or bowel movements, differing from that present just prior to operation. He had had no vomiting, constipation or tarry stools since the laparotomy. Roentgenologically the stomach was normal at this time except for a spot of slight deep tenderness in the general area of the old ulcer. We believe it may be assumed that in this patient the persisting symptoms are due to the chronic cholecystitis.

Ready displacement of the tumor by manipulation is of course a pathognomonic sign of bezoar if it is obtained, but we were impressed by the inconstancy of this when we were unable to demonstrate it in the second examination of the second patient, no doubt on account of the greater pain and rigidity present at this time. The absence of diarrhea in these two instances differs from the symptoms as usually given. The disappearance of the ulcer and of the acute symptoms after removal of the phytobezoar in the second patient makes it seem probable that the food-ball had produced the ulcer. In one of three other patients with phytobezoar coming under our observation, evidence of an ulcer which had healed with a deep crater defect was found.* Incidentally, it might be mentioned that none of these three gave a history of hematemesis and none had diarrhea.

Diagnosis from the roentgen appearance has been made only occasionally, no doubt because the frequency of its occurrence has been underestimated and the presence of a malignant, inoperable tumor has been diagnosed, although the roentgen appearance of all five of these was characteristic. The importance of differentiation is obvious and in view of the instances here reported attention is called to the fact that occurrence of gastric hemorrhage, lack of mobility of the tumor and association with ulcerative lesions are not inconsistent with a diagnosis of bezoar. Throughout a considerable portion of the United States the persimmon is found and undoubtedly many phytobezoars are wrongly diagnosed.

SUMMARY

1. Two instances of phytobezoar are reported with roentgen findings. Both patients suffered from severe gastric hemorrhage, a symptom to which but scanty reference is made in the literature.

2. Bezoar should be considered in the differential diagnosis of any intragastric tumor and benefit of exploratory operation given in the event that doubt as to diagnosis remains.

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[For Remainder of References see p. 569.]

* Report of this will be made by Dr. Wynn S. Owen.

NEW INSTRUMENTS

AUTO BLOOD TRANSFUSION APPARATUS

NELSON W. CORNELL, M.D.

NEW YORK

THE benefits from the use of the blood of a patient in the form of an auto blood transfusion are well known and have been described by Farrar.[1] The following apparatus was designed with two purposes in mind; first, the collection, citration, straining, and administration of the patient's own blood in the quickest possible time; secondly, to carry out this act with the least possible chance of any contamination of the aspirated blood.

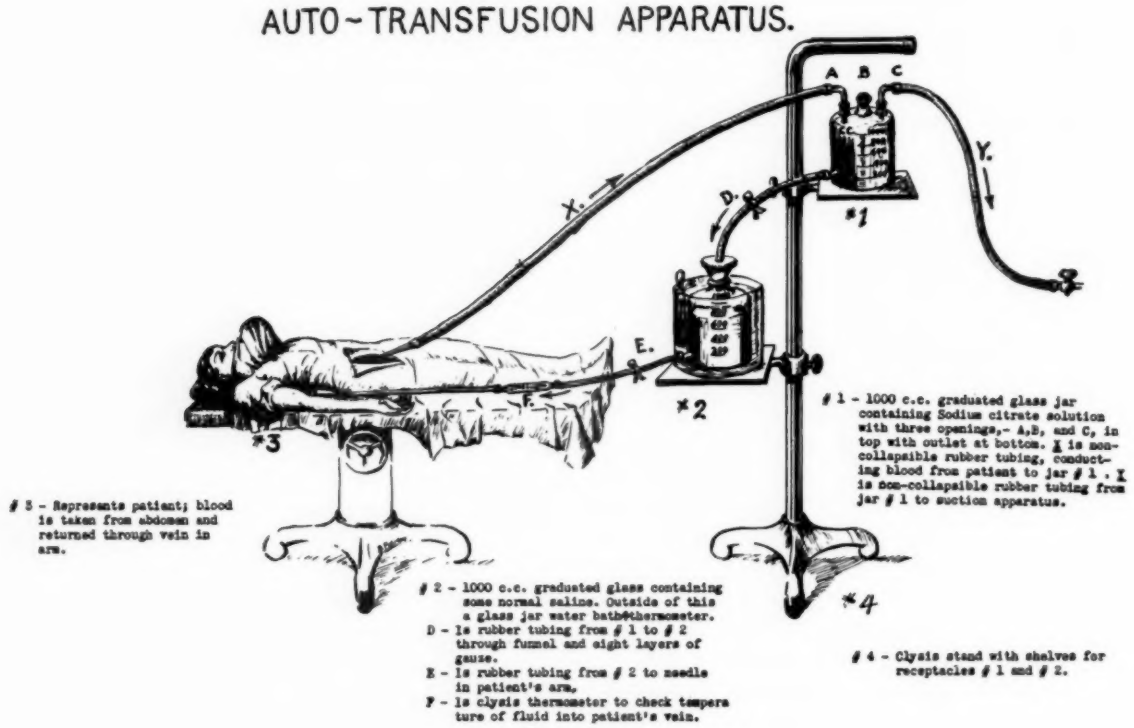
AUTO-TRANSFUSION APPARATUS.
A B C
X.
Y.
D.
#1
E.
#2
#3
F.
#4
1 - 1000 c.c. graduated glass jar containing Sodium citrate solution with three openings,- A,B, and C, in top with outlet at bottom. X is non-collapsible rubber tubing, conducting blood from patient to jar # 1 . Y is non-collapsible rubber tubing from jar # 1 to suction apparatus.
2 - 1000 c.c. graduated glass containing some normal saline. Outside of this a glass jar water bath&thermometer.
D - Is rubber tubing from # 1 to # 2 through funnel and eight layers of gauze.
E - Is rubber tubing from # 2 to needle in patient's arm.
F - Is clysis thermometer to check tempera ture of fluid into patient's vein.
3 - Represents patient; blood is taken from abdomen and returned through vein in arm.
4 - Clysis stand with shelves for receptacles # 1 and # 2.

FIG. 1.

Apparatus: This consists of a 1000 c.c. graduated glass jar (1) with three openings in the top and an outlet at the bottom; another 1000 c.c. graduated glass jar (2) which has one opening at the top and an outlet at the bottom which goes out through the side of the water bath, as in Figure 1; approximately 16 feet of ½ inch non-collapsible rubber tubing; two thermometers, one for the water bath, and the other a clysis thermometer to be inserted into the tubing conducting the blood into the patient's vein. These portions of the apparatus described can be made to individual specifications very cheaply. The

[1] Farrar, L. K. P. Auto-blood transfusion in gynecology. *Surg., Gynec. Obst.*, 49: 454–461, 1929.

clysis stand is usually found in any hospital, and the apparatus may be arranged on the stand, as illustrated.

Procedure: Ten cubic centimeters of sterile 2 per cent sodium citrate solution are used in every 100 c.c. of blood which are drawn into the graduated glass jar No. 1. Usually 50 c.c. of sodium citrate, which will take care of 500 c.c. of aspirated blood, are placed in graduated glass jar No. 1. If more blood is obtained, more citrate solution can be added through opening B by simply removing the cork. The blood is aspirated from the abdomen through the ordinary suction tip by means of a suction apparatus which is found in any well-equipped operating room. The agitation of the blood in glass jar No. 1 is brought about by the negative pressure caused by the suction, so that the citrate solution and blood are thoroughly mixed without the container being touched. When all the blood has been aspirated into the graduated jar No. 1, the clip D controlling the tube leading to glass jar No. 2 is released, and the blood allowed to flow by gravity through a sterile funnel covered by eight to ten layers of sterile gauze moistened with saline solution. (Dr. Farrar in her paper recommends twenty thicknesses of sterile gauze.) The mouth of the funnel is covered over by a sterile perforated watch glass, the rubber tubing running through the perforation in the center of the watch glass.

The 1000 c.c. graduated glass jar No. 2 is surrounded by a glass water bath which keeps the temperature between 105° and 110° F. In the graduated glass jar No. 2 there have already been placed 200 to 300 c.c. of sterile normal saline solution with which the citrated blood mixes as it is strained from glass jar No. 1. The clip E is then released and the mixture of blood citrate solution and normal saline solution is allowed to flow through the tubing and through the thermometer F into the patient's median basilic vein. The thermometer F rechecks the temperature of the solution as it is flowing into the patient's vein.

Advantages of this Apparatus: (1) It is an entirely closed apparatus, so that there is no necessity of the blood being handled or exposed to contamination, as there might well be where it is shifted from one open receptacle to another. (2) The blood can be rapidly collected, citrated, filtered, and re-injected into the patient's own veins. (3) The water bath about the graduated glass jar No. 2, together with a double-checking thermometer system, insures the necessary temperature at which the fluid should be given intravenously.

CONCLUSION

This apparatus has been used by the writer in a number of cases, and has been found to work perfectly from a mechanical and practical point of view.

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* Continued from p. 567.

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EDITORIAL

ANNIVERSARY

THIS issue of the JOURNAL completes six years of our editorship. During the past six years many changes have occurred. The first three years we were in the chair were years of plenty. We were finishing an era, although we did not know it at the time. The last three years have been years of want, a struggle of the survival of the fittest. The last few months have made us feel we are returning to good times again.

From 1928 to 1931 the circulation of the JOURNAL trebled. From 100 pages a month the size was increased to over 200 pages a month. Many issues were upwards of 250 pages. There have been several outstanding numbers: the Spinal Anesthesia number; the Gastro-Enterological number; the Minor Surgery number; the Matas Birthday number; the Southern Surgical Society numbers, etc. In addition, three supplementary volumes have been included: Livingston's The Peritoneum, Pollack and Davis' Peripheral Nerve Injuries and Cutting's Principles of Preoperative and Postoperative Treatment. These, at their conclusion, have been republished as books.

During the depression the Publishers have not ordered us to cut down or scimp one bit. They have constantly urged us to add to the quality and worth of the material. On their part they have kept the JOURNAL at its high standard of make-up.

We feel times are on the mend and one straw, alone, indicates this prediction. Since summer the circulation department

informs us several hundred new subscriptions have been added to the circulation. Subscribers who were forced by economic conditions to drop their subscriptions are resubscribing. All in all, the future is reassuring.

We have splendid material on hand for 1934. The January number, alone, may give an idea of what our readers can expect. For example, in the January number, among many worthy articles, will appear the following: Principles Involved in the Treatment of Carcinoma Affecting the Organs Located in the Male and Female Pelvis, by Robert C. Coffey; Clinical Value of Experimental Ureteral Transplantation, by Thomas J. Kirwin; Actinomycosis of the Subphrenic Space, by Amos M. Graves and Alton Ochsner; Presacral Dermoids, by Frank H. Lahey and Edwin B. Eckerson; Present-day Treatment of Colonic Cancer, by Fred W. Rankin; Large Malignant Tumor (Sarcoma) of the Uterus with Multiple Bone Metastases Successfully Treated by Hysterectomy and Coley's Toxins, by David Blum and William B. Coley; Myofascitis, by Fred H. Albee; Notes on a Series of Spinal Cord Tumors, by Francis C. Grant; Liver Deaths and Their Prevention, by Samuel Weiss.

We wish to take this opportunity to extend to our readers our thanks for their many kind letters and valuable suggestions. Even to those who wrote letters of complaint let us say they were not read and passed by lightly. And to the Publishers may we acknowledge our appreciation for their fairness and interest, and for their help over many hard places in the road.

T. S. W.

NOTICE

THE American Association for the Study of Goiter, for the fifth time, offers Three Hundred Dollars ($300.00) as a first award, and two honorable mentions for the best essays based upon original research work on any phase of goiter presented at their annual meeting in Cleveland, Ohio, June 7–9, 1934. It is hoped this will stimulate valuable research work, especially in regard to the basic cause of goiter.

Competing manuscripts must be in English, and submitted to the Corresponding Secretary, J. R. Yung, M.D., 670 Cherry St., Terre Haute, Ind., U. S. A., not later than April 1, 1934. Manuscripts received after this date will be held for the next year or returned at the author's request.

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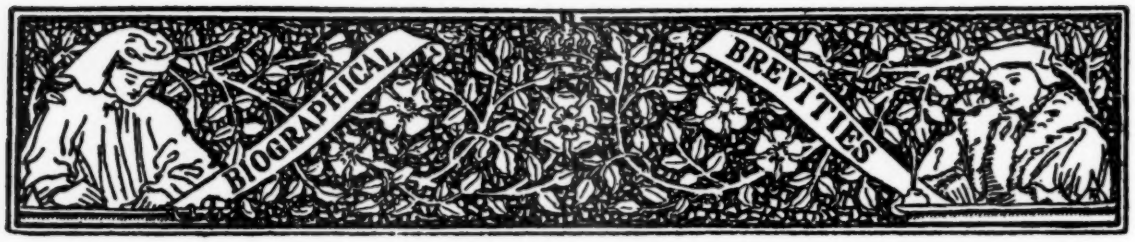

AMERICAN PHYSICIANS

DANIEL DRAKE

ONE of the most picturesque medical figures of the early West was Daniel Drake. He was born in New Jersey in 1785. When he was a child his family moved to Kentucky and there among the pioneers he spent his early years in direst poverty. His struggles for an education are epic. Without money, family or influential friends he overcame every obstacle and reached the topmost peaks of his profession.

Drake became a pupil of William Goforth, the "pioneer of Jennerian vaccination in the West," and his diploma was made out and signed by his teacher. It was the first medical diploma issued west of the Alleghanies. Drake started in practice. He never graduated from a recognized school of medicine, but the University of Pennsylvania in 1815 conferred upon him an academic degree. He was restless, combative. "He changed his locality as a teacher no less than seven times during his life, and two important medical faculties, the Medical College of Ohio (1821) and the Medical Department of Cincinnati College (1835) were founded by him." Drake founded the *Western Journal of the Medical and Physical Sciences*. It existed from 1827 to 1838, and in its time was the leading journal in the west. In its numbers appeared his writing on Medical Education, one of which has become famous. This essay appeared as a reprint in 1832.

In 1841 Drake published an account of a local disorder known as "the trembles" or milk sickness. He described an epidemic of cholera which appeared in Cincinnati in 1832. In 1831 he wrote a number of papers on the evils of city life. Three years later he wrote an essay of mesmerism, while his Moral Defects in Medical Students appeared in 1847. As a result of three decades of labor he wrote a great work on "Diseases of the Interior Valley of North America" (1850–54). A second volume appeared after his death. Garrison says that there "was nothing like this book in literature unless it might be Hippocrates on Airs, Waters, and Places. . . . " In 1810 he wrote on the Climate and Diseases of Cincinnati, and in 1822 a "Narrative of the Rise and Fall of the Medical College of Ohio." Today these are among the rarest of Americana.

Drake was always well dressed. He was an interesting lecturer and had a "splendid voice." He was kind, gentle, detested vulgarity and coarseness. He had a poetic bent and wrote creditable verse.

He did a great work in creating worthy medical teaching in Cincinnati.

Daniel Drake died in 1852 in his sixty-eighth year.

T. S. W.

DANIEL DRAKE

[1785-1852]

The American Journal of Surgery
N.S. Vol. XXII, December, 1933

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

MORE LIVE FISHES IMPACTED IN THE THROATS OF MEN

By E. W. Gudger

NEW YORK CITY

As bibliographer of fish literature, I am always on the lookout for unusual phenomena wherein fishes are concerned. Years ago my attention was called to the curious accident indicated in the title to this article. Various data were collected and in 1926 I published a paper bringing together in extensive quotations 20 cases of live fishes impacted in the food and air passages of men. I had at this time checked all the volumes then issued of the catalogue of the Surgeon General's Library, and as a consequence said in my article: "It is believed that all known accounts are included."

But it is impossible at the first historical survey of a subject to get "all known accounts." This was proved when on checking the *Index Medicus* and the *Quarterly Cumulative Index* to 1927, I found 11 additional accounts of this curious and interesting fatality. These we worked up and published in 1927. But this did not prove to be the end, for in checking the medical and ichthyological literature up to date (1933), three more accounts have been found. Further, four newspaper reports have come in (three authenticated beyond question) from a correspondent in the Far East. These accounts are cited to bring to date our knowledge of this remarkable accident. The cases are reported by countries.

FRANCE

In the *British Medical Journal*, 1863, there is a brief note of a fish-in-the-throat accident occurring at Toulon, France. However, this is but a repercussion of the account by Drs. Arland and Cobbold treated in my first article (1926).

PARAGUAY

Theodore Roosevelt in his "Through the Brazilian Wilderness"* records that in southern Brazil a man, whose hands were full, put the head of a vicious piranha in his mouth. The fish thereupon bit the man's tongue and nearly caused his death.

Fifteen years later, L. C. Beadle, writing of fishing in the nearby Paraguayan Chaco, states that:

> When each fish is caught it is killed in a somewhat barbarous fashion. The head is put into the man's mouth and is bitten through. The fish is then thrown ashore or placed in a string bag, which is often carried for fish or

*N. Y., 1914, p. 52.

game. The smallest fish, such as *Callichthys*, are often killed in this way, and it is on record that on one occasion a small *Callichthys* took a jump down a man's throat, and choking him put an end to the unfortunate fisherman.

NIGERIA—WEST AFRICA

In the same year a similar accident was reported from Nigeria, West Africa, by Dr. N. A. Dyce Sharp, as follows:

A few days ago a native woman was catching fish in the river with a basket net, and while transferring the small fry from the net she put one between her lips, her hands being full. The fish apparently gave a jump, and a moment later the woman was seen to fall, struggling to get her breath. Her friends at once opened her mouth and endeavoured to remove the obstruction; they failed, and after five minutes all her convulsive movements ceased. The body was at once brought to the mortuary, with a request for a necropsy, only afterwards did I discover that this was designed to determine whether she was a witch herself or whether she was bewitched, and here I failed! On removing the larynx and as much of the pharynx as possible, I found a small fish, 3¼ in. long, wedged firmly down the trachea, with the snout lying transversely between the vocal cords. Minute spines on the dorsal surface and at the border of the gills effectually prevented its extraction upwards, and, indeed, the tail had already been pulled off by the victim's friends. As the width of its head was about 23 mm. and the breadth about 17 mm., the larynx was most completely and exactly occluded. This is well shown in the preserved specimen, a smaller or a larger fish could not have caused so complete an obstruction.

Of the 31 cases previously reported, 15 are from India and the countries of the Indo-Chinese Peninsula, and of the 15 phenomena 13 were caused by that fish best known to us as the Indian "climbing perch," *Anabas scandens*. Of the following four cases from India and Siam, three are chargeable to this same fish.

INDIA

The following account was clipped from the Calcutta *Statesman* of July 24, 1927, and sent to me by the correspondent referred to. It fits in with other like accounts from India. The *Koi* is the "climbing perch."

A strange story is told in connection with the death on Friday evening at the Medical College Hospital, Calcutta, of Netai Charan Das, a Bengali boy, eleven years of age. It is stated that the lad caught a *Koi* fish in a submerged paddy [rice] field near his village. On his way home, he is stated to have held the fish between his teeth in order to have his hands free to climb over a fence which lay in his path. While negotiating the obstruction, the lad fell forward and the fish slipped down his throat and got stuck in his gullet. He was immediately removed to the Bagerhat sub-divisional hospital and from there brought down to the Medical College where he died the same evening.

Dr. Hugh M. Smith, Fisheries Adviser to the Siamese Government, sent me in 1926 data on a native Siamese choked by an *Anabas*. Recently he sent me three other authenticated accounts from Siam. The first is from the *Bangkok Times* of April 12 and 13, 1932:

A boy of thirteen lost his life on Sunday at Phra Khanong [a part of Bangkok] through having a *pla-mob* fish in his mouth. It wriggled down his throat and suffocated him.

In connection with the boy who died as the result of accidentally swallowing a *pla-mob* fish at Phra Khanong, his mother hastily buried his remains without first waiting for the authorities to examine the same. She is now held in custody pending further investigations.

The fish was the "climbing perch," one of the commonest market fishes in Siam, found in rice fields, bayous and backwaters. It is very hardy, active and long-lived, and hence is brought in alive to the markets.

The fish concerned in the second account sent by Dr. Smith was a fresh-water sole of the genus *Cynoglossus*. Here follows a translation of the account from *Siam Rasbdra Daily News*, a Bangkok paper of October 16, 1932.

On the 11th of October Nai Tongsook [a farmer] went fishing and caught various fishes

which were taken home and kept alive in a wooden tub of water. The man covered the bucket with a board which was held in place by a stone, and then went off to attend to other business. When his wife had nursed the child, she took a vessel and went to get water. While the mother was away the child went to the bucket where the fishes were kept, lifted the stone and cover, and got into the tub where she tried to seize the fishes with her hands. After a time she caught a small *pla lin ma* and put it head first in her mouth. Because of the movements of the fish and its mucous covering, it slipped into her throat and interfered with her breathing. This happened at 4 P.M. Some time later the parents came and discovered the child's condition, but did not know what to do, and she died of asphyxiation at 3 A.M.

This case happened in Tambon on Nong Srisa Kwai, Ampur of Nakon Luang, Changwad of Ayuthia. This district is very remote and no physician was available, so the child was left to die under very distressing circumstances.

Another account sent in by Dr. Smith, is an abstract from the *Siam Rasbdra Daily News* of January 9, 1933:

A boy named Fiem, about eleven years old, was left in the care of an elder brother while the parents went to work in the fields, distant about one day's walk. The residence of the family was in Tambol Sai Mai, in the Changwad of Patoom Dhani, Monthon of Ayudhia. On January 6, 1933, the boy took a water buffalo to pasture in a field, and, to pass the time, went fishing in a shallow swamp. About 11 A.M. he caught a small *pla mor* [*Anabas testudineus*] and, putting it in his mouth, ran happily to show it to his brother. Before he reached the house, the fish slipped into the boy's throat. Crying piteously, the boy sought his brother, and opened his mouth and pointed his finger to show what was the matter. The brother called on a neighbor to assist him in removing the fish, but they were unable to do anything. He then went to the *nai ampur* (mayor), who saw the boy and advised his brother to take him to the Central Hospital in Bangkok.

The brother and two other relatives took the boy by train to Bangkok and reached the hospital at 6 o'clock P.M., and a physician immediately removed the fish, which was still alive. The boy was very weak and died about ten minutes later.

Of this practice Dr. Smith writes that "the mouth is not an uncommon place in which adults and children in Siam hold fishes while rearranging their lines or when both hands are occupied in any way." *Anabas* has its head parts, especially the gill covers, beset with spines. When the fish is once impacted in the throat, these and the sharp dorsal spines make it almost impossible for the fish to be withdrawn.

These accounts from my files bring the history of this remarkable accident up to 1933. However, as this is a not infrequent happening, especially in the Far East, it is probable that other cases will later be put on record.

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BOOK REVIEWS

INFECTIONS OF THE HAND. A Guide to the Surgical Treatment of Acute and Chronic Suppurative Processes in the Fingers, Hand and Forearm. By Allen B. Kanavel, M.D. Ed. 6, Phila., Lea & Febiger, 1933.

That a sixth edition of this book has been called for is not only a tribute to the book and to the author but also to the surgical profession. This is a monograph of the highest order and the fact that it has taken its place among the big sellers is a very interesting comment on the improving standards of surgery. Dr. Kanavel's book has become and will remain a standard on this subject. It is perfectly astounding to note the amount of practical hard sense that is combined here with a tremendous amount of scientific research. This is one of the essential volumes for every surgeon's five foot bookshelf.

THE 1933 YEAR BOOK OF RADIOLOGY. Diagnosis, Edited by Charles A. Waters, M.D. and Therapeutics, Edited by Ira I. Kaplan, M.D. Chicago, Year Book Publishers, Inc., 1933.

The 1933 Year Book of Radiology, the second volume of the series, has been published promptly, a fact upon which both editors and publishers are to be congratulated. The material in this volume is well selected. The book is larger than last year's in both text and illustrations.

According to the Introduction "difficulties with regard to publication and the associated necessity of an increase in price in a time of universally reduced purchasing power induced us to omit many originally selected articles which otherwise would have been included." It is perhaps unfortunate that the editors were not able to include everything they wished to but it must be admitted that the material included is worthwhile and it would seem that little of value has been omitted. A splendid author and subject index make for easy reference.

Radiology now plays a part in every medical specialty and it is important for every physician to be posted on the new developments. It is to be hoped that the response of the profession will be such as to warrant a continuation of this publication for many years to come.

MANUEL DE RADIODIAGNOSTIC CLINIQUE. By R. Ledoux-Lebard. Paris, Masson and Cie, 1933.

A two volume manual of radiology of over 1000 pages by Dr. Ledoux-Lebard may be considered an event in roentgen literature. The author not only has a reputation as a clinician but also as a physicist and the value of this combination in roentgenology is evidenced on every page of this splendid production. There are 1143 illustrations, most of them splendidly reproduced and a large percentage of them original. The work is divided into 40 chapters making eight "books" which cover all the divisions of roentgenology, beginning with a Book of 60 pages on the "Production and Interpretation of Roentgenological Images." The chapter on the digestive tract is one of the largest in the book and one of the most valuable.

There is a bibliography at the end of each Book which makes no attempt at completeness but which is remarkably practical and contains references to American and English as well as Continental literature. No roentgenologist who can read French should be without this book.

MANUAL OF UROLOGY. By R. M. LeComte, M.D. Baltimore, William Wood & Co., 1933.

Dr. LeComte has written a short Manual of Urology for the use of medical students. He points out that the "work sets forth only generally recognized conclusions, without extended discussions, case reports or reference . . . it is intended to give the beginner a ground work of fundamentals on which to build a larger structure to be gained during his internship, postgraduate studies, and practice."

The author has succeeded in carrying out his intentions and it remains to be seen whether other teachers will find in this book just the material they want for their courses. There is no question but that the shorter textbook is an absolute necessity in the present over-crowded medical curriculum. It is to be

feared, however, that there are too many differences of opinion to warrant the acceptance of any condensed volume by any large number of teachers. It is likely, therefore, that out of the present needs of medical education will grow an even more voluminous literature than in the past consisting, however, of shorter volumes each containing the material which the author feels is essential. Sooner or later out of this mass of material will come some classic short volumes as classic larger works have appeared in the past.

Surgical Anatomy. By Grant Massie, M.D., M.S. (Lond.), F.R.C.S. (Eng.). Ed. 2, Phila., Lea & Febiger, 1933.

This is the second edition of a practical, condensed and conventional Surgical Anatomy. For students, this book may have much value and it will answer many questions for the practicing surgeon who will however usually require a larger and more complete work.

Diet and Dental Health. By Milton T. Hanke. Univ. Chicago Press, 1933.

This book has a broader interest than its title indicates. While several chapters are devoted to the question of dental changes through diet, the entire work is a thorough discussion of modern dietetics and metabolism. An unusual opportunity for research has been afforded the author through the cooperation of the Chicago Dental Research Club and the Sprague Memorial Institute. The California Fruit Growers Exchange have contributed financial assistance. The author feels that his researches have proved "that the average American diet is somewhat deficient in Vitamin c and that this deficiency may be a factor in dental as well as in other diseases." Three hundred and forty-one children at Mooseheart, Illinois, were offered as a test group for the data which are contained in over sixty pages of tables as the appendix. There are about 150 pages of text matter, each of them brim full of information on actual clinical and laboratory research. There are numerous splendid illustrations, both colored and black, and a seven page bibliography. While invaluable to research workers the book will also be found of practical interest and value to the dental practitioner and to the practicing physician and surgeon.

Ten Years of Obstetrics and Gynecology in Private Practice. By John L. Rothrock, M.D. N. Y., Paul B. Hoeber, Inc., 1933.

This is a book any physician who does general practice or specializes in obstetrics and gynecology will want to read in place of the latest novel. Not only will he read it for information and so add to his knowledge but he will find the pages are absorbing and have a lively interest for him. For, in the main, it is an account of ten years' experiences of a busy man in the field of obstetrics and gynecology. One will not always agree with the author's conclusions. This is well. We found ourself temporarily closing the book at times and thinking what we would do in a situation that confronted the doctor. Often we applauded his judgment, at other times we could not see with him eye to eye.

The book is divided into two parts. Part I deals with Obstetrics and relates the author's experiences and beliefs in about every obstetrical complication possible. Part II covers Gynecology and considers the more common disorders. The whole work is profusely illustrated with case reports.

All in all it is a book one will read and not glance through. When one finishes reading it he feels well repaid for the time spent between its covers.

Obstetrics and Gynecology. Ed. by Arthur Hale Curtis, M.D. Vol. 3. Phila., W. B. Saunders Co. 1933.

The third and final volume of this solid work is at hand.

The contents of the volume are: Displacements and Relaxations, Disturbances of Function, The Endocrines in Gynecology and Obstetrics, Special Diseases and Important Complexes, Other Gynecological and Symptom Complexes, Special Topics. Under each of these headings many subjects are considered. We cannot discuss each author or his topic. We particularly liked the opening chapter on Displacements. It would be advantageous for all physicians to read The Endocrines in Gynecology and Obstetrics, while the closing chapter, Special Topics, covers such subjects as The Early Months of Pregnancy from a Gynecological Aspect, Urinary Tract Problems in Gynecology, Radiotherapy, Blood Transfusion, Preoperative Problems, Anesthesia,

and Operative Management and Postoperative Care. We have omitted several other topics which are a part of this chapter.

Volume III rounds out a splendid work. There are 1664 illustrations. There is a small volume for a General Index which one will find valuable as a time saver.

If one had to select one work on obstetrics and gynecology and no other I cannot see how he could go amiss in selecting this work. To our mind it is the best published to date. Doctor Curtis should view his work with pride and have that inner satisfaction that his task has been well done.

BOOKS RECEIVED

All books received by THE AMERICAN JOURNAL OF SURGERY are listed in this column as soon as possible after their receipt and this must be considered as adequate acknowledgment. Books that the Editor considers of special interest to our readers will be reviewed in a later issue.

AMERIKA UND DER MEDIZIN. By Henry E. Sigerist. Leipzig, Thieme, 1933.

ARACHNOIDITIS SPINALES ADHÉSIVES (LES). By Dem. Paulian and D. Turnesco. Paris, Masson, 1933.

ARTHRODÉSES DANS LA COXALGIE (LES). By Paul Bufnoir. Paris, Masson, 1933.

BEHIND THE DOCTOR. By Logan Clendenning. M.D. N. Y., Knopf, 1933.

EMBOLIE (DIE). By Sigurd Frey. Leipzig. Thieme, 1933.

ENTWICKLUNG DER AUGENHEILKUNDE IM 18. UND 19. JAHRHUNDERT. By Alfred Bader. Basel, Schwabe, 1933.

FRACTUREN UND LUXATIONEN. By Georg Magnus Ed. 2, Berlin, Springer, 1933.

GREAT DOCTORS (THE). By Henry E. Sigerist, N. Y., Norton, 1933.

LUFT- UND FETTEMBOLIE (DIE). By Siegfried Hoffheinz. Stuttgart, Enke, 1933.

NEW FEET FOR OLD. By Martin Hiss, M.D. Garden City, N. Y., Doubleday, Doran, 1933.

OBSTETRICS AND GYNECOLOGY, VOL. 3. ED. by Arthur Hall Curtis, M.D. Phila., Saunders, 1933.

ORTHOPÄDISCHE GYMNASTIK. By Georg Hohmann and Lina Stumpf. Leipzig, Thieme, 1933.

ROCKEFELLER FOUNDATION ANNUAL REPORT, 1932 (THE). N. Y., Rockefeller Found., 1932.

RÖNTGENDIAGNOSTIK DER KNOCHEN- UND GELENKKRANKHEITEN. By Robert Kienbock. Vienna, Urban and Schwarzenberg, 1933.

ACUTE INTESTINAL OBSTRUCTION

MONROE A. McIVER, M.D., F.A.C.S.

TWELFTH INSTALLMENT (CONCLUSION)

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Chapter XXXII

THEORIES AS TO CAUSE OF DEATH (Continued)

Toxemia in Obstruction (Continued)

Absorption of the Toxin. *The Mucosa.* It may be considered an established fact that no absorption of a toxic substance can take place through a normal mucosa.* In other words, if the most virulent toxic fluid from an obstructed loop be placed in the normal intestine of another animal, no symptoms follow.†[2–4] This narrows the question to a consideration of what changes take place under conditions of intestinal obstruction which permit the poison to be absorbed. The most reasonable explanation is that the mucosa is so damaged or destroyed that it is permeable to the toxin from the lumen, or that after the barrier of epithelial cells is broken down the toxin may be actually formed in the intestinal wall itself, probably by the action of bacteria or by autolytic changes.

That degenerative changes frequently occur in the intestinal mucosa under conditions of obstruction and are the important factors in absorption has been stressed by many investigators.[2,8–11] These changes are usually attributed to the fact that the circulation to the bowel is damaged by distention. Since increased intraintestinal pressure is important in causing damage to the circulation and has also been considered to

* It is also true that if an obstructed loop be formed and toxic intestinal fluid placed in it at the same time, the onset of symptoms and death is not hastened.[1]

† Guillaume[5] is one of the few who question this, and he brings no real evidence in favor of a contrary view. The clinical observation (stressed by Guillaume) that a patient may go into collapse after being relieved from an obstruction does not prove that this occurred because the toxic material had passed into the normal bowel below the point of obstruction and had been there absorbed. As pointed out by Brooks et al.,[6] there are other explanations of this observation, among which may be mentioned the possibility of increased absorption from the already damaged mucosa; and in the case of strangulations there may be a puddling of blood in the dilated intestinal vessels after relief of the strangulation. Elman[7] suggests that when even a simple obstruction is suddenly released or a distended intestine quickly decompressed, great loss of blood and fluid into the splanchnic area may take place and produce serious symptoms.

play a direct rôle in causing absorption, it will be discussed in more detail.

Intraintestinal Pressure. As already stated, an increase in intraintestinal pressure has been supposed to influence absorption in two ways: indirectly by interfering with the circulation[8,9] and so producing injury to the mucosa; and directly by forcing the toxin into the absorbing channels.[1,2,12]

A number of experiments have been carried out to show the effect of increased pressure upon the circulation of the bowel. Van Zwalenburg[13] in 1907 placed an electric light in the intestine and observed under the microscope the effect on the circulation in the bowel wall of gradually increasing the pressure in the intestine by introducing water through a canula. He found that the circulation was markedly affected by high pressure.*

Gatch et al.[14] and Dragstedt et al.[15] have also studied the effect of distention on the circulation of the blood and found that the flow was diminished when the pressure in the intestine was increased. Dragstedt considered that interference caused by distention was greatest in the duodenum and least in the colon. Van Beuren's studies[10,16] of the pathological changes produced in the bowel by distention are also important: briefly these changes consisted of areas of infarction, necrosis and at times perforation, occurring for the most part on the antimesenteric border of the intestine. Burget et al.[17,18] found that animals with closed intestinal loops could be kept alive almost indefinitely if overdistention of the loop were prevented by aspirating the contents; if the pressure within the loop were allowed to increase, the symptoms of obstruction would immediately return.

In regard to the second point, namely, that an increase in intraintestinal pressure directly affects the absorption, experiments were carried out in 1924 by Stone et al.[12] and in 1927 by

* At 30 mm. of mercury some capillary streams were stopped. It required 130 mm. before all the circulation stopped; but as Van Zwalenburg pointed out, venous flow was probably retarded at slight increase of pressure.

Owings et al.[19] in order to determine the relation of increased pressure to absorption. Stone believed pressure was an important factor, not because it injured the mucosa and so indirectly permitted absorption of toxic substances, but because its presence directly caused abnormal absorption, presumably by forcing the toxic material into the absorbing channels. Murphy and Brooks[1] had previously advanced the view that increased intraintestinal pressure favored absorption and made practical use of it in one of their experiments; they believed, however, that there was an injury to the mucosa before absorption could take place. Owings et al.[19] made measurements of the intraintestinal pressure both under normal conditions and following obstructions: they found it considerably elevated during obstruction.*

Dissemination of Toxins. Granted that the toxin has passed the barrier imposed by the mucosa, there are three ways in which general dissemination might take place: the toxin might be taken up by the blood stream; it might be carried by the lymphatics; or it might diffuse into the general peritoneal cavity and be absorbed from that source. One might therefore look for the toxin in the blood, the lymph or the peritoneal fluid.

Blood Stream. The assumption has often been made that absorption takes place by the blood stream. The theory is plausible, but there is no important evidence, either for or against it. Experiments[4,20,21] showing that the blood of animals dying of intestinal obstruction is not toxic when injected into another animal prove nothing beyond that fact; the blood of animals poisoned in another manner may also be non-toxic when transferred to another animal.[3] On the other hand, the work of Sugito[22] and Scholefield[23] on the toxicity for mice of the blood of animals with obstruction is not convincing.

* It was, however, considerably below the high pressure created by Van Zwalenburg,[13] Gatch,[14] and others. It is possible that some other factor besides increased intraintestinal pressure affects the capillary circulation of the obstructed bowel: perhaps the permeability of the mucosa may be increased by the irritative action of the intestinal fluid so that a direct toxic action on the capillaries can take place.

Lymphatic Channel. Most of the adherents of the theory that there is an absorption of toxin from an obstructed intestine assume that absorption occurs both by way of the blood stream and by way of the lymphatics. Certain workers, however, have especially stressed the importance of the latter route. Murphy and Vincent[2] thought the onset of toxic symptoms was checked by the ligation of the lymphatic channels in the mesentery; their experiments were, however, not adequately controlled.

Murphy and Brooks[1] also collected material from the thoracic ducts of animals in whom obstructed loops with occluded veins had been formed, and from animals with obstructed loops in which the toxic content was under pressure. The collected fluid was found, in certain instances but not always, to be toxic on injection into a normal animal. Objections can be made to these experiments: the fluid was kept over night before injection into the normal animal; and in the control experiments slight symptoms (vomiting) were shown on the injection of thoracic duct fluid from an animal that had no obstruction. They are, however, interesting experiments and it would be most desirable to have the work confirmed.*

Morton,[24] using colloidal silver which has a selective staining action for lymphatics, was not able to demonstrate any staining of the thoracic lymphatics unless an actual perforation of the loops occurred.

Peritoneal Cavity as a Source of Absorption. Under conditions of obstruction there is usually an increase of free fluid in the peritoneal cavity. This is particularly true where there is an interference with the venous circulation. A toxin from an obstructed loop might diffuse into this fluid and then be taken up by the abdominal lymphatics and blood vessels. This possibility has been tested chiefly with the peritoneal fluid after producing interference with the circulation of the bowel (Fig. 58†). No toxic substance, however, can be detected in

* McIver and White were not able to confirm this, working with obstructed loops in cats. However, the amount of material collected was relatively small.

† Figure 58 appeared in the October installment, p. 173.

the peritoneal fluid[1,4,25] by injecting it intravenously into normal animals even when large quantities are used.

Although there is no direct evidence, there is considerable indirect evidence that diffusion of a toxin into the peritoneal cavity in presence of strangulation takes place. In 1812 Travers,[26] in studying the cause of death in strangulated hernia, drew a coil of intestine out of the abdomen and closed the wound around it in such a way as to strangulate the blood supply. The animal lived six days; the strangulated gut had sloughed and a fecal fistula had been established. Two other animals in which an internal strangulation was produced died on the second and third days.* White and McIver† also found that where a loop of intestine with its venous supply obstructed was brought outside of the peritoneal cavity the animal lived as long as though only a simple obstruction existed. McIver, White and Lawson[25] showed that if a twenty-four-hour strangulated loop were suspended in salt solution in the incubator for a few hours the salt solution surrounding the loop became toxic to normal animals. Foster and Hausler[28] concluded that placing short strangulated loops in a rubber bag (in order to isolate them from the peritoneal cavity) prolonged the life of the animal.

Studies on Absorption of other Substances, under Conditions of Obstruction. Since it is so difficult to obtain direct evidence of the absorption of the toxin, indirect evidence has been sought by studying the absorption of various other substances under conditions of obstruction. The gist of those experiments is that the absorption of most substances when placed in the obstructed intestine seems to be diminished rather than increased. Braun and Boruttau[29] after testing the absorption of strychnine from an obstructed loop came to the conclusion that the absorption of this drug was definitely

* This author recommended that in strangulated hernia where the bowel does not appear to be viable, an opening should be made in the intestine and the intestine allowed to drain. His contention is that under these conditions (i.e., strangulation outside of the peritoneal cavity) the patient dies of obstruction, not from intestinal gangrene.

† Experiments reported in article by White and Fender.[27]

diminished. Enderlen and Hotz[30] later determined the absorption rates of solutions of sodium chloride and glucose, and found that these also were decreased in the presence of obstruction. More recently Palma,[31] using phenolsulphonphthalein, came to the same conclusion.

Various types of dyes have also been used in studies on absorption. Morton[24] in his studies on absorption under conditions of obstruction worked with two classes of dyes: brilliant vital red and trypan blue, representing dyes of large molecular structure; and congo red, cloth red and congo blue representing dyes with smaller molecules. These dyes were placed in obstructed loops but could not be demonstrated in the vessels or in the wall of the living intestine.

The experiments of Hettwer and Kriz[32,33] on the absorption of protein from ligated intestinal loops are of interest and introduce a new method that may throw considerable light on the problem. These workers sensitized guinea-pigs to horse serum and found that they could produce anaphylactic reactions by introducing serum into ligated intestinal loops. This was shown to occur more readily when short rather than long loops were employed and they considered that the increased pressure in the short loops was an important factor in absorption. The experiments are of particular interest in that they furnish direct evidence of the absorption of foreign protein from an obstructed loop.

In conclusion it may be said that although these numerous experiments on the subject of absorption from the intestine under conditions of obstruction have furnished a great deal of valuable information, there is no conclusive evidence on the mechanism of absorption or the pathways by which general dissemination of the toxin occurs.

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Chapter XXXIII

THEORIES AS TO CAUSE OF DEATH (Continued)

The Theory that Death Is Due to Dehydration

The fact that dehydration appears as a frequent and severe complication of intestinal obstruction has been known for a long time.*[1,2] It remained, however, for Hartwell and Hoguet[3] in 1912 to formulate the definite theory that death in high intestinal obstruction is due to dehydration brought about by the loss of the digestive secretions. Neither they nor more recent advocates of this theory, however, have thought that the death in all types of intestinal obstruction could be explained on this basis. The application of the theory has been clearly limited from the beginning to simple obstructions. This fact is emphasized, since at times there has been misunderstanding on this point.

It is well to examine in some detail the important evidence brought forward by Hartwell and Hoguet in favor of their views. These workers found that animals dying from simple high obstruction seldom showed any noteworthy organic change in the intestine above the obstruction.† This led them

* Leichtenstern wrote in 1876: "The change of the features to the form of the facies hippocratica, the sinking of the eyes, the diminished turgescence of the skin, the aphonia, and the dryness of the skin, are due in part to the diminished absorption of water in consequence of the violent vomiting and to the withdrawal of water from the blood and tissues by the profuse sweats which usually occur simultaneously."

Nothnagel (1904) says: "This hippocratic face is, however, due to another factor present in this disease—namely, the great loss of water from the tissues, which is directly due to the profuse vomiting and the attacks of profuse perspiration that occasionally supervene in occlusion of the bowel. The loss of water from the tissues in addition causes wasting of the skin, dryness of the tongue, torturing thirst, and aphonia, conditions which are all occasionally seen in these cases. Leichtenstern has calculated the amount of water lost from the tissues, and his investigation shows that, together with the concentration of the blood, there is relative increase in the percentage of hemoglobin. The loss of water may be very considerable and may amount to 24 per cent of the volume of the blood."

† It was quite otherwise in the case of low obstruction. Here they often found extensive changes in the mucosa (which they attributed largely to circulatory disturbances following distention), and in these instances they could not prolong life by treatment with salt solution. They therefore concluded that when these degenerative changes

to doubt that death resulted from the absorption of a toxin. In seeking some other explanation they were struck by the large amounts of fluid lost by the animals in vomiting; and by a loss of weight (2.2 kg., for example, in three and one-half days) so rapid that it could only be accounted for on the basis of loss of water. These findings led them to restore to these animals the fluid lost, using physiological saline solution. They found that whereas untreated animals died in a few days (three to ten days), the animals treated by administration of normal salt solution, in excess of that lost in the vomitus,* lived three or more weeks, and the animals sacrificed at the expiration of that period gave the impression that they would have lived almost indefinitely if the treatment had been continued. The results of their experiments were so striking that they were led to advance the view that death in simple high obstruction is usually due to dehydration.

The full significance of this really remarkable discovery was not wholly appreciated at the time, largely because attention was focussed upon the theory of toxic absorption and also because a great deal of the experimental work was being carried out with closed duodenal loops, a complicated type of obstruction which is not materially benefited by the administration of salt solution.

The views of Hartwell and Hoguet were supported in 1913 by Wilkie[6] who stated very clearly that "simple obstruction high in the intestine differs from that lower down, chiefly in the great loss of intestinal secretions by vomiting in the former, compared with the latter, where the secretions are re-absorbed above the obstruction." This investigator obstructed the

in the mucosa occurred, whether in low or in high obstruction, death was due to the absorption of a toxin.[3,4]

* Whether the fluid is vomited or is retained in the stomach or upper intestinal segment above the obstruction, it is lost to the animals. A most striking example is found in the rabbit: this animal is not able to vomit, and under conditions of obstruction[5] the stomach acts as a reservoir in which large volumes of fluid collect, the animal showing the low blood chlorides and other findings of dehydration. Hartwell, Hoguet and Beekman[4] recognized the importance of the loss to the body of fluid accumulated in the stomach and intestines.

duodenum by means of a ligature and placed a rubber tube drain above this; he found that animals so prepared lost as much as one-third of their body weight in the thirty-six hours that they lived. In 1923 Haden and Orr published the first of a series of papers[7–10] in which they reported their important findings on the changes occurring in the blood chemistry following intestinal obstruction. These changes were, chiefly, a fall in the blood chlorides, an increase in the non-protein nitrogen, and a rise in the carbon dioxide combining power of the plasma. These authors also completely confirmed the observations of Hartwell and Hoguet on the efficacy of salt solution in prolonging the life of animals with high obstruction; and further showed that other solutions, although isotonic, are not efficacious in prolonging the life of the animals.* Gamble and McIver[5,12] also stressed the importance of sodium and chloride ions, and regarded their loss as largely responsible for the fatal effects of simple high obstruction. For while agreeing with Hartwell and Hoguet that dehydration was responsible for the death of the animals, they considered that dehydration† was dependent on the loss of the sodium and chloride ions. (See "Mechanism of Dehydration."‡)

Opponents of the view that dehydration is the most important factor in death from high intestinal obstruction cite certain experiments bearing directly on the water content of tissues. Draper[16] in 1914 measured the water content of the liver, kidney, muscle and other parenchymatous tissues under normal conditions and following fasting, salivation by pilocar-

* These authors at first thought that the blood chlorides were low because the chloride ion combined with and neutralized some toxic substance. They later modified this view.[11]

† Some writers, feeling that the term "dehydration" should properly refer only to the loss of water, have used the term "dehydration and dechlorination" to describe the conditions found after upper intestinal obstruction, i.e., decrease in body fluids and a low concentration of chloride in the blood. This, however, is also incomplete; for, although it is more difficult to demonstrate, a great loss of sodium as well as of chloride occurs. Indeed, Gamble and Ross have offered evidence that the loss of sodium is more important than that of chloride. It would seem simpler and better to use the old terminology but to employ it with a broader meaning.

‡ This appeared in the May installment, p. 490.

pine and duodenal obstruction. He found the water loss to be about 10 per cent in all three conditions. The fasting and salivated animals showed no signs of debility, while the animals with duodenal obstruction died. Whipple and co-workers[17] reported somewhat similar experiments. They found that dogs, dehydrated by means of drugs, showed no evidence of intoxication and recovered their weight very promptly when given food and water. Ingvaldsen and co-workers[18] measured the water content of spleen, muscle and liver following the production of high and low obstruction in dogs and found that there was only slight lowering of the water content of these tissues.

These various experiments were interpreted as showing that dehydration played no important rôle as a cause of death. Two criticisms may be made of this line of reasoning. In the first place, the loss of fluids produced by the use of pilocarpine is not comparable with the dehydration caused by loss of the digestive secretions, because sodium and chloride ions are not lost to such an extent. In the second place, the water content of the parenchymatous tissues such as muscle and liver does not furnish an accurate estimate of the degree of dehydration. As has already been pointed out, the most extensive loss of water occurs not from richly cellular tissue but from the interstitial spaces, particularly the subcutaneous tissue. This might be expected to be true on theoretical grounds (see Fig. 37*), and certain experiments[5] have indicated that such is actually the case.

The cause of death in simple high obstruction may be summarized by saying that it can be adequately explained on the basis of the loss of the digestive secretions and the secondary changes that follow.

Theory that the Cause of Death Is Anemia of the Vital Centers Resulting from Circulatory Disturbances in the Splanchnic Area

More than twenty years ago, Braun and Boruttau[19] and Wilkie[6] came to the conclusion that their work did not support

* Figure 37 appeared in the May installment, p. 491.

the view that death was due to the absorption of a toxin.* Braun and Boruttau were especially impressed by the fact that according to their experiments absorption instead of being increased under conditions of obstruction was actually decreased; and Wilkie was not able to produce toxic symptoms by injection of a Berkefeld filtrate of loop contents or expressed juice from the wall of a strangulated gut. Wilkie further found that animals with strangulation of long loops of intestine died within a few hours with symptoms of shock, a finding which has been more recently confirmed by Foster[20] and others. Foster and co-workers considered that the time elapsing before death was too short for the development of a toxemia. Braun and Boruttau and Wilkie considered that the most outstanding finding in patients dying of obstruction was the accumulation of fluid throughout the splanchnic area, in the engorged blood vessels, in the intestines above the obstruction, and in the peritoneal cavity itself. This drainage of fluid into the splanchnic area decreases the circulatory blood volume, thus lowering the efficiency of the circulation in general and of the cerebral circulation in particular. As Wilkie stated it, "splanchnic paresis with depletion of the systemic circulation is the main factor in producing the symptom complex of acute intestinal obstruction." More recently, Elman and co-workers,[21,22] doubting that absorption of a toxin is the cause of death in simple low obstruction, have also come to believe that possibly the stagnation of blood and fluid in the splanchnic area may lead to peripheral circulatory failure and shock in this type of obstruction.

There are many points that may be mentioned in support of the theory of splanchnic paresis. The splanchnic area, when the vessels are dilated, is capable of acting as an enormous reservoir for blood: as an example of this may be cited the noticeable drop in blood pressure following section of the splanchnic nerves. The engorgement of the vessels above even

* In simple high obstruction Wilkie considered that the loss of the digestive secretions in vomiting was the important factor. (See theory of dehydration.)

a simple obstruction is often a striking feature of the picture presented by these cases at operation; and a cyanotic tinge of the obstructed intestine, indicating stagnation in the capillaries, is also often noticeable. Rapid loss of body fluids (as has been fully discussed in the section on dehydration) follows the great outpouring of fluids into the lumen of the gut. All these facts make the theory a plausible one; and, furthermore, since these findings may be noted, in varying degrees, in almost all cases of obstruction, it is probable that they play some rôle in most instances, and is at least possible that in certain types of obstruction, where the death has not been adequately explained on the basis of other factors, they may play the determining part.

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Chapter XXXIV

Conclusions as to Cause of Death in Different Types of Obstruction

The widely divergent results that follow different types of experimental obstruction and the distinctive features shown by the various types of obstruction encountered in humans, have made it increasingly obvious that no one theory will explain the cause of death in all varieties of intestinal obstruction. It is also clear from the analysis of the experimental work in the preceding chapters that (as shown in Table xxviii*) while certain types of obstruction produced in the lower animals are comparable to obstructions occurring in humans, others are not.

In this chapter the author draws certain conclusions, on the basis of the evidence at hand, as to the cause of death in the different types of intestinal obstruction. It must be realized that these conclusions are only tentative and in many instances the evidence on which they are based is meager and at times self contradictory.

Cause of Death in Simple High Obstruction. In simple high obstruction the most likely explanation of the symptoms and death of the animals seems to me to be dehydration, due to the loss of the digestive secretions in vomiting. The term "dehydration" must include a loss not merely of water but also of the essential sodium and chloride ions. The severe secondary changes that occur in the body fluids and tissues as a result of the loss of these essential materials have been discussed under the section on dehydration (p. 490).

Cause of Death in Simple Low Obstruction of the Small Intestine. Simple low obstruction in humans occurs much more commonly than high obstruction and is therefore more important. Unfortunately, experimental obstructions of this type in laboratory animals do not always closely

* Table xxviii appeared in the Oct. installment, p. 178.

parallel the disease in humans, and therefore the data on which to base an opinion as to the cause of death is somewhat limited. An outstanding feature at operation or autopsy is usually marked distention of the intestinal coils above the obstruction; the tension is only partially relieved by drainage back into the stomach and by vomiting, and an increase in intraintestinal pressure occurs. This pressure undoubtedly interferes with the capillary circulation and is a factor in starting the degenerative process in the bowel wall which may eventually result in permeability to a toxin. It is possible that vascular stasis and exudation in the splanchnic area are of greater importance than is generally recognized, and warrant further study.

The part played by dehydration in this type of obstruction varies considerably. Where vomiting is profuse, the dehydration is often severe. It cannot alone, however, explain the symptoms and death; for the survival period in animals or humans cannot usually be materially lengthened by the administration of salt solution.

Cause of Death in Simple Obstruction of the Colon. The lower animals with simple obstruction of the colon may live for considerable periods of time without symptoms and eventually die of inanition. In humans, also, the disease is not so quickly fatal as that resulting from obstructions of the small intestine. In neglected cases distention of the cecum may become so great that the blood supply is cut off, areas of gangrene and perforation with resultant peritonitis occur, and death results.

At times the picture presented by the patient is not unlike that found in low obstructions of the ileum; and at operation or autopsy the whole of the small intestine as well as the colon proximal to the obstruction may be distended and filled with the characteristic foul fluid. The death in these cases is presumably the same as that in low obstructions of the ileum.

Cause of Death in Obstructions Characterized by Interference with the Mesenteric Blood Supply. Interference with the mesenteric circulation to the bowel adds

a new factor to the causes of death already discussed, and is of great clinical importance because of its frequency and seriousness. As examples may be mentioned the various types of strangulation, volvulus, intussusception or mesenteric thrombosis.

The symptoms and death in obstructions characterized by interference with the mesenteric circulation can, it appears to me, be best explained on the basis of a toxemia caused by the action of bacteria multiplying in the necrosing intestinal wall. According to this conception the toxemia would be comparable to that arising from gangrenous tissue located anywhere in the body, modified only by the fact that the intestines are suspended in a large serous cavity where the temperature is most favorable for the rapid multiplication of bacteria and for the disintegration of tissue, and from which absorption readily takes place. Degenerative changes in the bowel wall may go on to a point where a swiftly spreading peritonitis occurs, with or without macroscopic perforation.

It must be realized that the degree of interference with the circulation varies greatly in different instances. It is obvious that where the interference with the mesenteric circulation is at a minimum, the whole picture more nearly approaches that of simple obstruction, the overlapping of types producing numerous variations of the clinical picture. The symptoms are not likely to be greatly modified by the location of this type of obstruction. It is sometimes said that, just as in simple obstruction, the higher the obstruction the more fulminating the symptoms. There is no good evidence in the literature for this statement; on the face of it, it seems unlikely. The suddenness and completeness of the strangulation are probably the most important facts in influencing the course of the disease.

Reduction of blood volume by the transudate of plasma and fluids into the involved bowel and by stasis in the capillaries and mesenteric vessels may play a rôle in the symptoms and death. The importance of this factor probably increases in proportion to the length of bowel infarcted.

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